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# PYRILAMINE MALEATE IN THE TREATMENT OF PRURITIC DERMATOSES

RAYMOND C. V. ROBINSON, M.D.

Pyrilamine maleate, known commercially as Neo-antergan®, is one of a large series of compounds which have come to be known as antihistamines. The present communication deals with combined oral and local use of the drug in 184 patients with various dermatoses (Table I).

Antihistamine compounds may be divided into derivatives of 5 basic chemical structures. Neo-antergan (N-p-methoxybenzyl-$N^1$,$N^1$-dimethyl-N-a pyridylethylene diamine maleate) belongs to the group which has ethylene diamine as a base. It is available in tablets of 25 and 50 milligrams each, and has been incorporated in ointment bases for investigative purposes. The ointment used in the present series consists of 5 per cent of the drug in the following base:

| | |
|---|---|
| Carbowax 1500 | 0.5000 |
| Carbowax 4000 | 0.2500 |
| Propylene glycol | 0.2500 |
| | 1.0000 |

The only criteria for selection of patients were the presence of clinically apparent lesions and the subjective complaint of itching. Each patient was instructed to spread the ointment lightly on the affected parts 4 times daily and to take, at the time of each application, a one hundred and twenty-five milligram tablet of the drug. The latter dose, although somewhat smaller than is usually recommended, was decided on since it was felt that this amount might minimize possible side reactions without losing effectiveness to any great extent. Observations were made at weekly intervals unless shorter periods were indicated by the severeness of the condition.

The effects on pruritus ani and pruritus vulvae were particularly encouraging. In all 15 cases of pruritus vulvae with infiltration and leukoplakia tissues had softened and the leukoplakia had involuted at least 75 per cent by the end of the third week.

Materials used in this study were supplied by Merck Chemical Company and the Tarbonis Company.

TABLE 1

*184 Patients Treated with Pyrilamine Maleate Locally and Orally*

| DIAGNOSIS | NUMBER OF PATIENTS | GOOD RESULTS (1) | FAIR RESULTS (2) | FAILURES (3) | REACTIONS (4) |
|---|---|---|---|---|---|
| Pruritus vulvae with leukoplakia | 15 | 13 | 1 | | 1 |
| Pruritus vulvae without leukoplakia | 5 | 2 | | | 3 |
| Pruritus ani | 26 | 18 | 2 | 6 | |
| Dermatitis venenata (rhus) | 5 | | 1 | | 4 |
| Insect bites | 3 | | | | 3 |
| Contact dermatitis of hands | 16 | 10 | 2 | 4 | |
| Pityriasis rosea | 3 | 1 | | 2 | |
| Lupus erythematosus, chronic discoid | 3 | | | 3 | |
| Schamberg's disease | 1 | | | 1 | |
| Epidermophytid | 8 | | | 7 | 1 |
| Folliculitis | 8 | | | 8 | |
| Seborrheic dermatitis | 13 | 8 | 2 | 1 | 2 |
| Atopic dermatitis, chronic | 78 | 50 | 15 | 10 | 3 |

Good results: Relief of itching and 75 per cent or more improvement objectively during observation period.

Fair Results: Fifty per cent relief subjectively and/or objectively.

Failures: No improvement.

Reactions: Increase in itching, or objective lesions, or definite contact dermatitis.

Both objective and subjective improvement have been maintained in each case for 2 to 20 months. The degree of objective improvement, apparently not stressed in the literature as a result of treatment with similar antihistaminic compounds, was unexpected and has not yet been confirmed by biopsy. Further studies of this phase are planned. Subjective symptoms were alleviated after the first application of the ointment.

Of the 26 patients with pruritus ani, 18 received prompt relief and 2 noticed some improvement, although it was necessary to use roentgen therapy on both groups before the condition was controlled.

Four of 5 patients with dermatitis venenata (rhus) developed increased itching, burning and objective evidence of inflammation following treatment. One obtained partial relief of symptoms. An unfavorable reaction was also obtained in the 3 patients with insect bites.

A diagnosis of contact dermatitis of the hands was made in 16 patients. Two of these patients had had the condition for 2 or more months and were helped by Pyrilamine Maleate. Two patients had noticed the eruption for only a week or two and obtained slight relief.

One of 3 patients with pityriasis rosea seemed definitely improved but the condition was beginning to involute when first seen so that it is not certain that the course of the disease was materially influenced by the medication. There was no apparent effect in the other two.

No appreciable improvement was observed in 3 patients with chronic discoid lupus erythematosus, 1 with Schamberg's disease, 3 with epidermophytid and 3 with folliculitis.

TABLE II

*Comparison of Effects of Pyrilamine Maleate Locally and Orally with Roentgen-Radiation and/or Other Local Medication in the Treatment of Eczema*

| | NUMBER OF PATIENTS | | GOOD RESULTS (1) | | FAIR RESULTS (2) | | FAILURES (3) | |
|---|---|---|---|---|---|---|---|---|
| | Acute | Chronic | Acute | Chronic | Acute | Chronic | Acute | Chronic |
| Neo-antergan® | 13 | 45 | 2 | 32 | 3 | 12 | 8 | 1 |
| Roentgen-ray plus local medication* | 11 | 30 | 8 | 22 | 3 | 7 | 0 | 1 |
| Local medication without roentgen-ray | 7 | 10 | 4 | 4 | 2 | 3 | 1 | 3 |

1. Good results: Relief of itching and 75 per cent or more improvement objectively during observation period.
2. Fair results: Fifty per cent improvement subjectively and/or objectively.
3. Failure: No improvement or exacerbation of symptoms.

* 50 r weekly, unfiltered, at 90 K.V.P.

Maximum benefit was obtained in chronic and subacute eczema (78 with atopic dermatitis and 13 with seborrheic dermatitis, which will be discussed together). Eight of the 11 failures and all 5 local reactions occurred in patients suffering from acute exacerbations.

Table II compares the results in an identical number of patients treated with other modalities (roentgen or local preparations other than Pyrilamine Maleate).

From Table II it may be seen that roentgen radiation in addition to a variety of local medications, offers a better chance for good results in acute eczemas than does Pyrilamine Maleate alone. The roentgen radiation is at least as effective as this drug in the more chronic conditions. The combination of radiation and Pyrilamine Maleate has not been tried to any great extent in this study.

Seventeen patients in the control series did not receive any roentgen therapy for various reasons. Many had already received extensive treatment from roentgenologists or other dermatologists. In some of the older patients with senile skin changes, radiation was thought to be contra-indicated; in most of the acute cases roentgen radiation was withheld to be used only if local medications failed.

The experience gained in this study parallels much of the published work of investigators of other antihistaminics. Sulzberger, Baer and Levin (1), using Pyribenzamine ointment in a series of 16 patients with lichen simplex chronicus, found definite improvement in 8 and some improvement in 4 others. Shelmire (2) reported that 233 of 305 patients with pruritic dermatoses benefited from local use of Thephorin. Thirty-two of 56 patients observed by McGavach and his co-workers (3) had complete relief from itching with a 5 per cent Benadryl ointment.

## SUMMARY AND DISCUSSION

It is the opinion of the author that Pyrilamine Maleate ointment is as effective as other antihistamine-containing ointments (1–3) in the control of chronic eczematous eruptions. Adverse reactions were seen in 17 of the 185 patients treated, however, none occurred in those individuals with chronic eczematous conditions, pruritus ani or vulvae, where the preparation appeared to exert maximum beneficial effect.

The most significant observation in this study is the remarkable and unexpected objective improvement of pruritus vulvae with leukoplakia. This must be confirmed in a larger series of patients with histopathologic studies.

Four patients who suffered local reactions following application of the ointment were subsequently patch tested with the base and failed to give a positive test in 96 hours.

## CONCLUSION

Pyrilamine Maleate ointment is effective in the relief of chronic pruritic eczematous eruptions and in pruritus ani et vulvae. There is definite benefit subjectively and apparent reduction in the thickening of tissues and leukoplakia, which is frequently associated with chronic pruritus vulvae.

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## EDITORIAL

### THERE'S A DIVIDEND IN REHABILITATION

That the physically handicapped have entered the public's conscience can be attested by the reception given such motion pictures as *The Stratton Story*, *The Men*, *The Best Years of Our Lives*, and *Bright Victory*. The triumph of Helen Keller, the late President Roosevelt, Connie Boswell, and Johnny Ray, is known to school child and swing record enthusiast alike. In these instances of accomplishment—in spite of steel braces, hearing aid, or artificial limb—abilities have been demonstrated that transcend the skills of many who are whole-limbed and equipped with the normal range of senses.

With lesser known persons, the efforts of the specialist in rehabilitation have produced people capable of being employed, of raising families, and of contributing to society's advance. A study by the Civil Service Commission has demonstrated conclusively, that when handicapped workers are compared with physically fit employees in the same job (and statistical validation solidified the contention), they:

1. Are as good as, or better than able-bodied workers in both quality and quantity of work produced.
2. Do not change jobs so frequently, either voluntarily or as a result of managerial action.
3. Have fewer lost-time accidents.
4. Compare favorably with the unimpaired in absenteeism records.

Of what meaning are these findings to the physician? Wherein do these statements coincide with his daily routine of diagnosis, prognosis and treatment?

Those who care for patients with chronic or long protracted illnesses, or conditions where the body configuration or function will change, can plan the therapeutic regimen to include a full rehabilitative program. This implies not just a healing of the acute process, but a training toward self-sufficiency. It is what is left, not what has been damaged or destroyed, that makes for marketable skills in today's employment philosophy.

Further, these physicians who examine applicants for industrial positions can, with re-orientation of thinking toward skill-utilization and away from disqualification because of disability, aid industry in its procurement of sufficient manpower. This is necessary to maintain the production of both defense materiel and consumer goods. The time-frayed concept of meeting rigid physical standards to obtain work, must be replaced by the more logical matching of a man's somatic and emotional capacities with the physical and psychologic demands of the job. If his capacities to do, meet the requirements of the task, then he can perform that job irrespective of the associated disabilities.

Physicians have industrialists as patients. They can familiarize them with the efforts of the President's Committee on Employment of the Physically Handicapped —"hire the disabled, it's good business." Together, the employer and his medical adviser can add to the seven million so-called handicapped who are working today. Working, "because rehabilitation, training, counseling, and employment experts are considering the worker as a worker and the handicap as incidental."

*J. S. Felton, M.D.**

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# ACUTE APPENDICITIS

ALLAN H. MACHT, M.D. AND HOWARD M. KERN, M.D.

Since Reginald H. Fitz (1) of Boston first coined the term "appendicitis" in 1866, surgeons have been extremely interested in this disease process. With impetus from Thomas G. Morton (2) of Philadelphia, who first successfully removed the appendix in a planned operation in 1887, and Charles McBurney (3), who in 1889 described McBurney's point as the spot where localized pain and tenderness were usually found in cases of inflammation of the appendix, the diagnosis and treatment of acute appendicitis was established on a firm foundation. Even Today, the diagnosis of acute appendicitis still offers a challenge to every surgeon.

This study includes 892 cases that were operated on in Sinai Hospital, Baltimore, Maryland, with the preoperative diagnosis of acute appendicitis from January 1, 1948, to January 1, 1951. The accuracy of diagnosis, success of treatment, and pertinent data in the history and the physical examination of these patients were carefully reviewed. Included in this series of 892 cases are 87 cases of appendicitis having either generalized or localed peritonitis with abscess formation. There was no mortality. All diagnoses were verified by microscopic examination of the specimens and the only aspect left to the operating surgeon were his preoperative diagnosis and physical examination.

## Diagnosis

The preoperative diagnosis was confirmed by surgery in 576 cases. Thus, the surgeon made the correct diagnosis in about 2 out of 3 cases. In the males, the preoperative diagnosis was correct 73.53 per cent of the time, while in the female this was true in only 54.33 per cent of the cases. One hundred and eighty-four cases or approximately 20 per cent had no pathologic process demonstrable, and the pathologist reported "chronic appendicitis." In the remaining 132 cases, there was another abnormality present, either in the appendix or in the abdominal cavity, which could have caused the patients' signs and symptoms. If oxyurasis vermicularis infestation, lymphoid hyperplasia, and arteritis of the appendix can give signs and symptoms of acute appendicitis, then the preoperative diagnosis could be considered correct in 69.73 per cent of the cases.

## Age Incidence

The incidence of acute appendicitis in both the male and female (Table II) tends to be in the first 3 decades of life and gradually tapers off as age increases. In the age group that did not exceed 30, there were 403 cases of acute appendicitis or 69.96 per cent of the total cases in this series. The youngest was a 2 year old female and the oldest person a 79 year old male. Each of these patients had generalized peritonitis resulting from a ruptured appendix at the time of admission.

From the Department of Surgery, Sinai Hospital, Baltimore, Maryland.

## History

Most of the acute cases began with pain in the epigastrium gradually localizing in the right lower quadrant followed by nausea, vomiting and anorexia. However, in 15.97 per cent with acute appendicitis, the pain started initially in the right lower quadrant (Table III). Whereas in the cases of pathologic "chronic" appendicitis 40.76 per cent of the cases had pain beginning in the right lower quadrant as did 36.36 per cent of the cases with other lesions. There was no apparent difference in the acute and chronic cases of appendicitis in relation to gastrointestinal symptoms. However, about a quarter of the cases with other lesions were free of any gastrointestinal symptoms (Table III). In females, acute appendicitis occurred on any day of the menstrual cycle, with just as many cases arising during the period of menstrual flow as on any other day of the cycle. One case occurred 3 days post

TABLE I

1. Cases diagnosed pre-operatively as acute appendicitis

| | | | |
|---|---|---|---|
| Males | 476 | or | 53.36% |
| Females | 416 | or | 46.64% |
| Total | 892 | | 100.00% |

2. Pathologically "acute appendicitis"

| | | | |
|---|---|---|---|
| Males | 350 | or | 73.53% |
| Females | 226 | or | 54.33% |
| Total | 576 | | 64.58% |

3. Pathologically "chronic appendicitis" with no other pathologic process demonstrable

| | | | |
|---|---|---|---|
| Males | 82 | or | 17.23% |
| Females | 102 | or | 24.52% |
| Total | 184 | | 20.63% |

4. Other lesions found in the appendix or elsewhere in the abdomen

| | | | |
|---|---|---|---|
| Males | 44 | or | 9.24% |
| Females | 88 | or | 21.15% |
| Total | 132 | | 14.8 % |

TABLE II

*Incidence of Acute Appendicitis According to Age*

| AGE IN YEARS | MALES | FEMALES |
|---|---|---|
| 0–10 | 71 | 54 |
| 11–20 | 88 | 59 |
| 21–30 | 79 | 52 |
| 31–40 | 50 | 25 |
| 41–50 | 31 | 14 |
| 51–60 | 15 | 12 |
| 61–70 | 13 | 9 |
| 71–80 | 3 | 1 |

partum and 3 cases occurred during pregnancy. The time in the cycle midway between periods was of little help in differentiating the cases of acute appendicitis, because as many patients were operated on at this time, with the diagnosis being confirmed, as in any other part of the cycle.

It is interesting to note that despite an active campaign on the part of the medical profession in warning patients with abdominal pain not to take a laxative, 64 patients or 7.18 per cent took a cathartic after their abdominal pain had begun. It is appalling that in some instances, the cathartics were ordered by their physician. Twenty of these patients or 22.99 per cent had ruptured appendices (Table IV).

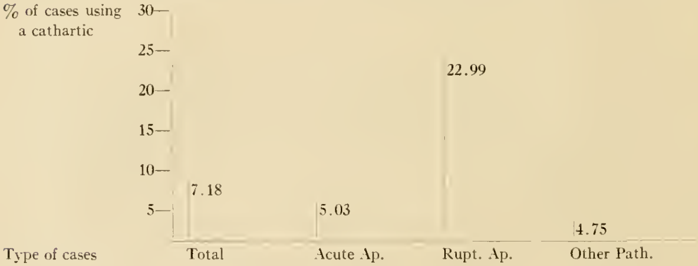
TABLE III

*A History Study*

| | ACUTE APPENDICITIS, 576 CASES | CHRONIC APPENDICITIS, 184 CASES | OTHER PATHOLOGY, 132 CASES | TOTAL, 892 CASES |
|---|---|---|---|---|
| Pain starting in the right lower quadrant | 92 or 15.97% | 75 or 40.76% | 48 or 36.36% | 215<br>24.10% |
| No gastrointestinal symptoms | 78 or 13.54% | 34 or 18.48% | 35 or 26.52% | 147<br>16.48% |

TABLE IV

*The Misuse of Cathartics in Patients with Abdominal Pain*



## Physical Examination

In this series 70.85 per cent of all cases entered the hospital or consulted a physician within 24 hours of the onset of symptoms, (Table V). However, 65.57 per cent of the ruptured appendicitis cases did not see a doctor until after 24 hours had elapsed. While 81.59 per cent of the acute appendicitis cases received medical attention within 24 hours, only 62.26 per cent having other disease processes or chronic appendicitis consulted a doctor within this period. Thus, it seems that the longer the patient waited to consult a physician, the better were his chances to have other or no pathology rather than an acute appendix if, of course, the appendix had not ruptured in the interval.

Most cases in this series showed tenderness, muscle spasm, and rebound tenderness on examination of the abdomen. A significant number of cases which did not prove subsequently to have acute appendicitis, often failed to show 1 or possibly 2 of these signs. All patients in this series had abdominal tenderness to palpation. However, 232 cases of 26.01 per cent of these examined had no discernible muscle spasm. While this occurred in 11.07 per cent of the patients with acute appendicitis, 59.78 per cent with chronic appendicitis and 48.48 per cent with other pathologic conditions showed absence of muscle spasm to abdominal palpation. Rebound tenderness was not elicited in about ⅓ of the total cases. In 17.19 per cent of the cases with acute appendicitis rebound tenderness was absent. However, about ¾ of the patients with chronic appendicitis and about ½ of the patients with other pathologic aspects did not show rebound tenderness. In 18.83 per cent of the cases only abdominal tenderness without muscle spasm or rebound tenderness was apparent. In this group only 5.38 per cent had an acute appendix. Chronic appendicitis occurred in 48.91 per cent of this group while 35.16 per cent had other pathology at the time of operation. Thus, while rebound tenderness or muscle spasm is present at least 82.81 per cent of the time in cases of acute appendicitis, it is present only in 51.52 per cent of patients without an acute appendix (Table VI).

TABLE V

*The Time Interval Before Consulting a Physician*

| TIME INTERVAL | ACUTE APPENDICITIS | CHRONIC APPENDICITIS OR OTHER PATHOLOGY | RUPTURED APPENDICITIS |
|---|---|---|---|
| 1–24 hours | 409 | 203 | 30 |
| 36 hours | 27 | 18 | 10 |
| 2 days | 35 | 33 | 25 |
| 3 days | 10 | 31 | 9 |
| 4 days | 4 | 13 | 3 |
| 5 days | 2 | 8 | 2 |
| 6 days | 0 | 2 | 5 |
| 1 week | 2 | 10 | 1 |
| 2 weeks | 0 | 8 | 2 |

TABLE VI

*The Physical Examination in These Cases*

| PHYSICAL SIGNS | ACUTE APPENDICITIS | CHRONIC APPENDICITIS | OTHER PATHOLOGIC ASPECTS | TOTAL |
|---|---|---|---|---|
| Abdominal tenderness | 576 or 100% | 184 or 100% | 132 or 100% | 892 or 100% |
| Muscle spasm | 518 or 89.93% | 74 or 40.22% | 68 or 51.52% | 660 or 73.99% |
| Rebound tenderness | 477 or 82.81% | 52 or 28.26% | 57 or 43.18% | 586 or 65.7% |
| Neither muscle spasm nor rebound tenderness | 31 or 5.38% | 90 or 48.91% | 47 or 35.61% | 168 or 18.8% |

## Surgery

Surgery was performed on all of the cases within a few hours after admission to the hospital. Whenever the diagnosis of acute appendicitis is made, the case is considered as a surgical emergency and is taken to the operating room as soon as possible. If dehydration is evident and the urine shows the presence of acetone bodies, surgery is delayed a few hours until intravenous fluids can be administered to overcome this condition as much as possible. The anesthetic administered varied depending on the age and condition of the patient. However, a majority of the patients received a sodium pentothal induction with various gas mixtures as the basic anesthetic. Spinal anesthesia was used in less than 5 per cent of the cases. In 81.84 per cent of patients, the abdomen was opened through a McBurney, muscle-splitting incision in the right lower quadrant. In 9.3 per cent of cases the next most common incision used, especially in females, was the right rectus muscle-splitting incision. The appendix was retrocecally located in 15.58 per cent of the patients. After the appendix had been removed, the stump was not inverted in 429 or 48.09 per cent of the cases. There was no morbidity from the appendiceal stump in either the inverted or non-inverted cases. The incisions were usually closed with chromic catgut except in the contaminated cases when stainless steel wire was used.

TABLE VII

*The Type of Incision Used*

| TYPE | MALE | FEMALE | TOTAL |
|---|---|---|---|
| McBurney muscle-splitting | 428 or 89.92% | 302 or 72.59% | 730 or 81.84% |
| Right trans-rectus | 28 or 5.88% | 55 or 13.22% | 83 or 9.3% |
| Paramedian | 7 or 1.47% | 32 or 7.69% | 39 or 4.36% |
| Pararectus | 9 | 15 | 24 |
| Transverse | 3 | 6 | 9 |
| Midline | 1 | 6 | 7 |

TABLE VIII

*The Number of Ruptured Appendices as Compared with Nonruptured Appendices*

| AGE IN YEARS | NUMBER OF ACUTE APPENDICES | NUMBER OF RUPTURED APPENDICES | PER CENT OF RUPTURED APPENDICES |
|---|---|---|---|
| 0–10 | 125 | 31 | 24.8 |
| 11–20 | 147 | 16 | 10.95 |
| 21–30 | 131 | 7 | 5.34 |
| 31–40 | 75 | 10 | 13.33 |
| 41–50 | 45 | 7 | 15.56 |
| 51–60 | 27 | 6 | 22.22 |
| 61–70 | 22 | 7 | 31.82 |
| 71–80 | 4 | 3 | 75.0 |
| Total | 576 | 87 | 15.1 |

## Ruptured Appendicitis

This report covers 87 cases with the pathologic diagnosis of a "ruptured" appendix or 9.75 per cent of the total number of cases operated upon or 15.1 per cent of the cases having acute appendicitis. Expressing this figure in a different way: in our series one out of every 6.5 cases of acute appendicitis was ruptured. The youngest child and the oldest patient encountered had ruptured appendices. A complete tabulation of these cases is given in Table VIII. It seems that this entity is more than common in the extremes of life.

The cases of ruptured appendicitis can be further subdivided into those with general peritonitis or those with the formation of a localized abscess. All the patients having perforations on the appendix were proved by microscopic examination. There were 60 cases or 68.9 per cent which had generalized peritonitis at the time of operation while the remaining 27 cases had localized abscess formation. In the last few years, most surgeons at Sinai Hospital placed drains within the peritoneal cavity when localized discreet abscesses were encountered during surgery. Usually patients with generalized peritonitis are drained by means of Penrose rubber tissue drains which are placed down to the peritoneal closure only (Table IX). It seems that in either instance a large percentage of cases have wound infections or develop pelvic abscesses.

TABLE IX

*The Incidence of Infection and Drainage in Cases of Ruptured Appendices*

| SITE OF DRAINS | CASES OF GENERALIZED PERITONITIS | | CASES OF LOCALIZED PERITONITIS | |
|---|---|---|---|---|
| | Infected | No infection | Infected | No infection |
| In peritoneal cavity | 9 | 7 | 7 | 8 |
| To peritoneal cavity | 9 | 29 | 0 | 12 |
| None used | 2 | 4 | 0 | 0 |
| Total | 20 | 40 | 7 | 20 |

TABLE X

*The Types of Complications in Various Cases*

| COMPLICATIONS | CASES OF ACUTE APPENDICITIS | CASES OF RUPTURED APPENDICITIS | OTHER CASES | TOTAL CASES |
|---|---|---|---|---|
| Wound infection | 8 | 23 | 2 | 33 |
| Pelvic Abscess | 0 | 7 | 0 | 7 |
| Ileus | 6 | 10 | 2 | 18 |
| Pneumonia | 2 | 4 | 5 | 11 |
| Atelectasis | 2 | 2 | 2 | 6 |
| Spinal headache | 2 | 0 | 1 | 2 |
| Hemorrhage from appendiceal artery | 1 | 1 | 0 | 2 |
| Hemiplegia | 1 | 0 | 0 | 1 |
| Hematoma of incision | 0 | 1 | 3 | 4 |
| Acute gastric dilatation | 0 | 1 | 0 | 1 |
| Dehissence | 0 | 0 | 1 | 1 |

## Complications

There were post-operative complications in 70 cases, 17 of which had multiple complications. Thus, there was an overall morbidity of 7.85 per cent without a mortality. In the group of patients with acute appendicitis, the morbidity was 3.47 per cent while those having chronic appendicitis with or without other pathologic aspects had a 4.43% morbidity. However, the rate of complications, for the cases of ruptured appendicitis was 41.38 per cent (Table X).

The average hospital stay for the 765 uncomplicated nonruptured cases, receiving surgery was 5.2 days. Patients with ruptured appendices stayed 8.3 days. When postoperative complications occur in patients having ruptured appendices, the average hospital stay is increased to 16.5 days. The longest single hospitalization in this series was 55 days. An average hospital stay of patients with nonruptured appendices who developed postoperative complications was 9.8 days.

TABLE XI

*The Hospital Stay of the Different Groups of Cases*

| DAYS | CASES OF UNCOMPLICATED APPENDICITIS | CASES OF COMPLICATED APPENDICITIS | CASES OF RUPTURED APPENDICITIS | CASES OF COMPLICATED RUPTURED APPENDICITIS |
|---|---|---|---|---|
| 2 | 8 | | | |
| 3 | 111 | | 1 | |
| 4 | 177 | | | |
| 5 | 182 | 2 | 5 | |
| 6 | 143 | 6 | 6 | 1 |
| 7 | 77 | 4 | 10 | 1 |
| 8 | 30 | 2 | 11 | 3 |
| 9 | 16 | 2 | 4 | 4 |
| 10 | 10 | 3 | 6 | 1 |
| 11 | 5 | 6 | 2 | 1 |
| 12 | 2 | 5 | 2 | 1 |
| 13–55 | 4 | 4 | 4 | 22 |
| Average stay in days.. | 5.2 | 9.8 | 8.3 | 16.5 |

### CONCLUSION AND SUMMARY

1. While the preoperative diagnosis of acute appendicitis was made in 892 cases, the diagnosis was correct in about 65 per cent. In females the surgeon was incorrect 50 per cent of the time. Whereas in males he was correct 66⅔ per cent of the time.

2. Although most of the cases of acute appendicitis occurred in the first 3 decades of life, it could have occurred at any age. The chance of rupture at the extremes of life was proportionately greater.

3. Gastrointestinal symptoms and the time of menstruation seemed to be of little help in making a diagnosis. When the pain began in the right lower quadrant instead of the typical epigastric location, the patient's chances of not having a lesion in the appendix seemed greater.

4. People still take cathartics and delay in consulting doctors despite the educa-

tional campaigns of the medical profession. These patients who waited and purged themselves had a higher incidence of ruptured appendices.

5. On physical examination, all patients had abdominal tenderness to palpation. However, muscle spasm or rebound tenderness was absent in at least 1/3 of the cases examined, and of this group about 50 per cent had lesions outside of the appendix. When both muscle spasm and rebound tenderness were absent, 82.14 per cent had lesions other than acute appendicitis.

6. The use of McBurney incisions and early surgery was the procedure of choice in most of our cases.

7. About 1 in every 6.5 cases of acute appendicitis had a ruptured appendix at the time of surgery. In the ruptured cases, drains within the abdominal cavity or to the peritoneum were used as a matter of choice. Only about 40 per cent of the other cases had postoperative complications. These complications, of course, added to the hospital stay, almost doubling hospitalization over the uncomplicated cases.

An attempt has been made to give the status of acute appendicitis at Sinai Hospital. So far, the morbidity rate has been about average but the mortality rate is far below average. The percentage of correct diagnoses has been presented. A study is also made to determine aspects of history and physical examinations which could possibly lead towards more correct diagnoses. The public still needs to be actively advised of the harm in taking cathartics and when the pain begins, the need of consulting a medical authority.

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# URINARY EXTRAVASATION*†

JOHN F. HOGAN, JR., M.D.

Generally speaking, this topic could embrace the broad field of conditions in which urine is found in the tissues about the urinary tract, i.e., from the kidney to the external urinary meatus.

The scope of this paper is confined to the extravasation of urine from the lower urinary tract. A few cases of extravasation from the urinary bladder will be mentioned, although this report, mainly, concerns periurethral phlegmon, or extravasion of urine from the urethra.

To understand the clinical course of this disease one must review the anatomy of the surrounding fascial layers present in the area.

Gustave Buck in treating a localized abscess at the base of the penis at the New York Hospital in 1848, was stimulated to do some anatomic work. He found that the penis had a distinct membranous sheath investing it—continuous above with the suspensory ligament and "perineal fascia" below and investing the corpora cavernosa and spongiosa penis.

Further description of this fascial layer, in light of present day knowledge, shows that it is attached to the inferior layer of the urogenital diaphragm and inferior rami of pubis and ischium. From this origin most workers (1, 2) agree that Buck's fascia proceeds upward involving the origins of the 3 components of the penis with their corresponding muscles. The separate compartments being—the bulb of the corpus spongiosum urethra with the bulbocavernosus muscle in the center, the corpora cavernosum penis with the ischio-cavernosus muscle of each side, in lateral compartments. The fascia continues up the shaft of the penis to the corona surrounding all 3 of these erectile tissue bodies. Tobin and Benjamin (1) have pointed out that a transverse septum of Buck's fascia separately encloses the corpus spongiosum urethra.

More recently Uhlenhuth (2) has disputed the fact that Buck's fascia immediately surrounds the ischio-cavernosus muscles. His work on dissecting cadavers shows that Colle's fascia gives off a major leaf in the perineum which expels a slip that passes deep to the bulbocavernosus muscle. This slip separates it from Buck's fascia and together with the major leaf of Colle's, forms a fascial compartment for the muscle. Uhlenhuth states further that a crural septum is also given off from the leaf of Colle's, which passes down adhering to crus "just caudal to the inferior urogenital fascia" and thus encloses the ischio-cavernosus muscle in separate fascial compartments. He claims that the Buck's fascia described in the perineum by Tobin is really Colle's and what Tobin terms albuginea is really Buck's fascia.

Colle's fascia takes origin from the posterior inferior border of the urogenital diaphragm and, laterally, (the inferior rami of pubis and ischium), and passes over the dorsal aspect of the superficial transverse perineal muscles. It travels down and forward under the skin of the perineum and scrotum forming a roof for the superficial urogenital pouch. Colle's fascia is continuous with the dartos of the scrotum and penile dartos. Further superiorly this layer is continuous with Scarpa's fascia of the anterior abdominal wall.

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* From the Department of Urology, Baltimore City Hospitals.

† Read before Baltimore Urological Society, March 15, 1951.

The above description, generally speaking, covers the anatomic features of Buck's and Colle's fascia necessary to understand the course of urine extravasated from the anterior urethra. For the membranous and posterior urethral lesions, one must discuss the triangular ligament or urogenital diaphragm.

Callender (3) has described the latter as "a semi-rigid musculo-membranous structure attached to the ischiopubis rami, and stretched tightly across the pubic arch. It separates the perineum from the pelvis and consists of two fascial layers between which are the deep transverse perineal muscles, the membranous urethra and its sphincter, Cowper's glands, the artery to the bulb, the internal pudendal vessels, and the dorsal nerve to the penis. The inferior or anterior fascia alone possesses any strength and blends with Colle's fascia. The superior or posterior layer of fascia is an undifferentiated fibrous structure, which really is an extension from the parietal pelvic fascia through the interlevator cleft." There are 3 main areas in which urethral rupture with secondary extravasation of urine may occur. If anterior to the urogenital diaphragm, it follows the layer between Buck's and Colle's fascia. Tobin (1) has shown that the majority of urethral ruptures and peri-urethral abscesses in general pass first into the corpus spongiosum. This readily accounts for any bacteremia, so often seen with the condition. From there it extends through Buck's fascia to the space between it and Colle's fascia. Because of the distribution of Colle's fascia, this would therefore be seen clinically in the perineum, scrotum, penis and anterior abdominal wall.

Rupture of the membranous urethra usually lacerates the delicate posterior fascia layer of the urogenital diaphragm, permitting extravasation posteriorly to the perivesical and retroperitoneal space.

Rupture in the posterior urethra, i.e., above the diaphragm, likewise invades the peri-vesicular and retroperitoneal tissues. In this type there is often extension to the ischio-rectal fossa.

It must be borne in mind, however, that the highly infected urine invading the tissues in peri-urethral phlegmon is usually capable of dissecting through fascial boundaries. Only early in the disease process may the extravasation be marked into one of the above stated areas.

In the present reported series, 6 cases showed infiltration of the tissues of the thigh. Also, 8 cases extended to the ischiorectal fossa from what was undoubtedly an anterior urethral extravasation in the beginning (see Table 1). Culp (4) has stated that late extravasation involving the penis, from the perineum and scrotum in the anterior variety, probably dissects between Buck's and Colle's fascia on the penis rather than under Buck's fascia. This is because of the tenacity of the latter structure to the underlying corpus spongiosum urethra. Scrotal extravasation is between the dartos (continuation of Colle's) and external spermatic fascia. In general then, any incision of infiltrated urine in the scrotum would not have to directly expose the testes, since these structures lie beneath the external spermatic fascia.

*Etiology:* The most frequent cause for periurethral phlegmon is urethral strictures, as will be seen in Table 2.

TABLE 1

*Objective Findings*

| EXTENT OF EXTRAVASATION | CASES | PER CENT |
|---|---|---|
| Perineal | 13 | 8.6 |
| Perineum<br>Scrotum<br>Penis<br>Abdominal wall | 45 | 30 |
| Perineum<br>Scrotum<br>Abdominal wall | 10 | 6.6 |
| Penis<br>Scrotum | 12 | 8 |
| Perivesical | 9 | 6 |
| Perineum<br>Scrotum | 14 | 9.33 |
| Scrotum<br>Penis<br>Abdominal wall | 20 | 13.3 |
| Scrotum<br>Abdominal wall | 7 | 4.6 |
| Penis | 1 | 6 |
| To Thigh | 6 | |
| Perineum<br>Scrotum<br>Penis | 13 | 8.6 |
| Scrotum | 5 | 3.3 |
| Penis<br>Abdominal wall | 1 | 6 |
| To ischio-rectal fossa | 8 | |

The chronically infected area of the stricture becomes invaded with the virulent organisms of the secondary urinary tract infection. This weakens the area of the urethra and by a combination of bacterial invasion and straining to void, perforation of the urethra is produced. Infected urine then passes out from the albuginea of the penis to the corpus spongiosum urethra. Fineston (5) believes that, in so doing, this process causes vascular thrombi and localized thrombophlebitis, which accelerates the

TABLE 2

*Etiology*

- A. Smith & Finton (BCH) 151 cases
  - 106 cases or 70%—Urethral strictures
  - 26 cases or 17%—Instrumentation
  - 7 cases or 5%—Trauma (external)
  - 12 cases or 8%—Miscellaneous
- B. Howland 60 cases
  - 49 cases—Strictures
  - 5 cases—Instrumentation
  - 4 cases—Trauma
  - 2 cases—Unknown
  - 1 case—Urethral calculus
- C. Present series (BCH) 150 cases
  - 95 cases (63.33%)—Urethral strictures
  - 37 cases (24.66%)—Instrumental (sounds, cystoscopes)
  - 5 cases (3.33%)—External trauma
  - 4 cases (2.66%)—Unknown
  - 9 cases (6%)—Miscellaneous including the following:
    - 2—Hernia repair with suture placed through bladder
    - 2—Extension from carcinoma of rectum
    - 1—Carcinoma of prostate
    - 1—Carcinoma of urethra
    - 1—Postoperative perineal prostatectomy with stricture
    - 1—Gangrene of bladder with intraperitoneal perforation
    - 1—Chemical burn of penis

infectious process. He also believes that, in those cases that continue to void, the action of the bulbo and ischiocavernosus muscles "squeezes" more urine into the tissues. This is accomplished by either directly breaking down Buck's fascial layer through bacterial invasion or, as often is the case in instrumental trauma, having an aperture broken through Buck's fascia as originally described above.

In the traumatic cases of urethral rupture, granting non-infected urine prior to the accident, a more anatomic extravasation should occur. If the trauma is not promptly treated by diverting the urinary stream, the same aspects of peri-urethral phlegmon can supervene in time, when the extravasated blood and urine become secondarily infected.

Table 2 gives the large preponderance of strictures as the etiologic agent and also some variety of other causes.

*Symptoms:* Subjectively, the patient usually states that he has had progressive narrowing of the urinary stream, attended by greater or lesser degree of dysuria, burning, and hesitancy. Frequently he will relate a history of past urethral dilatation. Often the precipitating event in the development of the condition is an alcoholic bout.

In the traumatic cases caused by instrumental perforation, local swelling, and pain with inability to void are seen. If occurring in the process of transurethral resection of the bladder neck, pain and swelling of the abdomen, with accompanied shock is seen.

TABLE 3

*General Information*

150 cases from 1935–1951

Sex—All males
Age—Youngest 8 years (traumatic)
Eldest 78 years

Color—White 43
Colored 107

A. There were two cases under 20 years
  (20–30 years)—18 cases
  (30–40 years)—20 cases
  (40–50 years)—32 cases
  (50–60 years)—35 cases
  (60–70 years)—20 cases
  (70–80 years)—21 cases
B. Duration of symptoms
  Shortest—4 hours
  Longest—12–14 days
    Average—3.26 days
C. NPN (Pre-sulfa)
  Fatal cases—with NPN reported—36 cases
    Average NPN—78.25 mgm%
  Living cases—with NPN reported—20 cases
    Average NPN—33.85 mgm%

In cases after chemotherapy no particular correlation existed between admission NPN and final outcome of case.

Objectively, one finds swelling, redness and warmth in the infected cases—corresponding to the area of urethra that is perforated. In the cases of phlegmonous infiltration, this rapidly progresses to gangrene of the superficial tissues, with accompanied slough later. In the traumatic cases the combination of blood with the extravasated urine will, naturally, give rise to a more ecchymotic appearance to the tissues.

The usual case of peri-urethral phlegmon presents itself 36 to 48 hours after the process has started (see Table 3). The patient is acutely ill, dehydrated, and often delirious as a result of uremia. In addition, he may be seriously acidotic, anemic and generally toxic. Often he is unable to give any history, and the first clue to the cause of his condition is gained when inspection of the genitalia reveals enormous swelling with patchy areas of gangrene.

The diagnosis is made from the history of urinary difficulty (if obtained) and calibration of the urethra, if not obtained. The general toxic state combined with local cellulitis should make chronic lymphedema easily differentiated. Lack of the same two factors, combined with clinical congestive heart failure should also eliminate confusion with genital edema as a counterpart of generalized anasarca.

Hinman and Tashiro (6) have written an excellent paper on the differential diagnosis of peri-urethral phlegmon and peri-rectal abscesses. They emphasize careful evaluation of the integrity of the lower urinary tract in any cases of ischio-rectal swellings, to prevent missing early treatment of peri-urethral phlegmon.

*Bacteriology:* Howland (7) has stressed routine cultures in these cases at the time of

TABLE 4

*Bacteriology*

| AUTHORS | BACTERIOLOGY | NO. OF CASES |
|---|---|---|
| Char, Shih, et al.—Chinese Medical Journal | Strept.<br>Staph. aureus | Most frequent<br>Next most frequent |
| Howland | Anaerobic strept. | 14 |
| | E. coli | 4 |
| | No growth | 4 |
| | staph. aureus<br>staph. albus<br>aerogenes<br>proteus | Miscellaneous |
| Smith and Finton BCH-1941 | Not enough cultures for specific comment | |
| Present series–cultures reported in only 36 cases | B. coli | 11 |
| | Staph. aureus | 6 |
| | Gamma strept. | 3 |
| | Proteus | 3 |
| | Anaerobic strept. | 2 |
| | Strept. fecalis | 2 |
| | Staph. albus | 2 |
| | Beta Hem. strept. | 2 |
| | Hem. staph. aureus | 2 |
| | No growth | 3 |

operation. His 14 cases of Anaerobic streptococcus infections point out clearly the need for this procedure (see Table 4).

Perhaps with modern anti-biotic and chemotherapeutic drugs, we rely too dependently on a satisfactory course postoperatively. To intelligently treat these cases, knowledge of the invading organism is necessary and may be life-saving in their postoperative course.

As Table 4 shows, our record of obtaining cultures is poor. In 1941 Smith and Finton (8) reported their series of cases and made a like plea for routine culturing.

## TREATMENT

### A. *Operative*

Everyone confronted with peri-urethral phlegmon agrees that prompt operative diversion of the urinary stream with adequate incision and drainage of all extravasated or phlegmonous areas, should be carried out.

As will be seen in Table 5, our method of urinary diversion has, in the overwhelming majority of cases, been performed by suprapubic cystostomy. It is likewise seen, that in comparable series at other institutions, external, i.e., perineal, urethrostomy is the procedure of choice, rather than suprapubic cystostomy.

TABLE 5

*Operative Treatment*

- A. Ockerblad—15 cases—1 death (6.6%)
  - 8 suprapubic cystostomy with I & D
  - 7 catheter drainage
    - (Most of latter were peri-urethral abscesses)
- B. Culp—18 cases—22.2% mortality
  - 12 perineal urethrostomy and I & D
  - 3 suprapubic cystostomy with I & D
  - 3 urethral catheterization with I & D
    - All these cases had extravasation to penis, scrotum, and perineum.
- C. Baker, et al
  - Pre-sulfa
  - 1. 100—External urethrostomy; I & D
    - 43% mortality
    - 13—Suprapubic cystostomy
      - 62% mortality
  - 2. With sulfa 112 patients
    - 102—External urethrostomy with multiple I & D
      - 39% mortality
    - 6—Suprapubic cystostomy; I & D
      - 3 died—50%
  - 3. With penicillin 25 cases
    - 21—Perineal urethrostomy; I & D
      - 20 lived—1 died
      - One patient had extravasation to axilla and was treated with external urethrostomy, I & D—lived
    - 2—Suprapubic cystostomy—1 died
- D. Riekert—Does external urethrostomy with I & D multiple
  - Post-sulfa 75 cases
    - 20% mortality—15 deaths
- E. Present series (BCH) 150 cases
  - 135—Suprapubic cystostomy
    - Multiple I & D
  - 4—Suprapubic cystostomy only
  - 4—I & D later Suprapubic cystostomy
  - 3—Urethral catheterization and I & D
  - 1—I and D only
  - 1—Urethral catheterization only
  - 1—Suprapubic cystoscopy; I & D later
  - 1—No treatment
- F. Char, Shih, et al 30 cases
  - 22—External urethrostomy; I & D
  - 4—I & D only
  - 2 Suprapubic cystostomy; I & D
    - 23% mortality

Riekert (9), series D on Table 5, summarizes the feeling of those who prefer this method of treatment as follows.

1. The urinary obstruction incident to the stricture is relieved and the fundamental cause of the infection is thereby corrected.

2. The infected peri-urethral tissue is provided with drainage.
3. The dependent position of drainage favors complete emptying of the bladder
4. The abdominal infiltration is superficial in the great majority of cases and suprapubic cystostomy performed through this infected tissue would introduce the virulent organisms into the deeper, uninfected tissue under the rectus sheath.

He also points out that the suprapubic sinus will not heal in these patients until adequate urethral dilatation and urethral catheter drainage has been instituted, in the event suprapubic cystostomy is used, and thus concludes that external urethrostomy will not prolong the postoperative course.

Baker (10), et al in their report of 272 cases at Cook County Hospital also prefers external urethrostomy to suprapubic cystostomy. In the post-sulfa group of 112 patients, they report 102 were treated by urethrostomy rather than suprapubic cystostomy. Their mortality was 39.2 per cent. In the post-penicillin series of 25 cases—1 death—they had 1 case with extensive extravasation—as far as the axilla. This case recovered through the use of multiple incisions and drainage, external urethrostomy and anti-biotics.

Certainly in all cases of bladder injury or extravasation from the posterior urethra, suprapubic cystostomy with drainage of the perivesical tissues would be indicated.

It is logical to be concerned about extensive perineal phlegmon treated by external urethrostomy. Fascial planes are not respected by this infectious process and extension to the peri-vesical area could take place. In such cases external urethrostomy would not provide adequate drainage.

Regardless of what choice of urinary diversion is employed, it is customary to make a generous incision and drainage of all affected tissues. These incisions should be made deeply enough to unroof Colle's fascia.

### *B. Supportive*

The supportive care of these patients concerns itself with sufficient intravenous fluids to combat dehydration and to treat uremia, if present. Glucose in water is generally preferable to saline because of danger of sodium retention in renal insufficiency.

Adequate blood should be given to combat the secondary anemia. Several advanced cases in this series were denied blood when it surely appeared mandatory from their charts. Multi-vitamins, and a high caloric diet are necessary. The local care of the operative wounds with Sitz baths, KMNO4 soaks and heat is helpful. Smith and Finton (8) have mentioned Zinc peroxide for local use in the anaerobic infections. The patients should be watched carefully for secondary hemorrhage from incised areas.

Drs. Toulson and Brantigan (11) advocated use of electro-excision of the impassable type stricture. They could often secure indwelling catheter drainage in badly scarred urethras by this method.

### *C. Specific supportive therapy*

Specific supportive therapy with sulfa drugs and anti-biotics has, of course, greatly changed the former aspects in this dreadful condition. As will be seen in Table 6, the

TABLE 6

*Mortality*

| TYPE OF TREATMENT | NO. OF CASES | NO. OF DEATHS | PER CENT |
|---|---|---|---|
| A. (After Riekert) | | | |
| Before sulfa and penicillin | | | |
| Campbell (1915–1929) | 135 | | 43 |
| Finestone (1930–1939) | 32 | | 50 |
| Smith and Finton (BCH) (1925–1940) | 133 | | 56 |
| Riekert (1932–1938) | 103 | | 38 |
| Post-sulfa and penicillin | | | |
| Beard (1938–1946) | 92 | | 25 |
| Riekert (1940–1946) | 75 | | 20 |
| B. Baker | | | |
| Pre-sulfa | 113 | | 50.4 |
| With sulfa | 112 | | 39.2 |
| With penicillin | 25 | | 16 |
| C. Present series | 150 | 73 | 48.66 |
| Pre-chemotherapy | 62 | 44 | 70.9 |
| Post-chemotherapy | 88 | 29 | 32.9 |
| After penicillin and sulfa | 25 | 3 | 12 |
| Streptomycin, penicillin, and sulfa | 9 | 0 | 0 |

immediate effect after the introduction of sulfa compounds, was reducing the mortality by half. Since penicillin, another drastic reduction has taken place.

Since employing streptomycin, penicillin and sulfadiazine or gantrisin, there have been no deaths from this disease in the Baltimore City hospitals. The last patient to die of perineal phlegmon in the Baltimore City Hospitals was on January 8, 1948.

### COMPLICATIONS

The 4 most common complications of the disease are listed in Table 7.

The first postoperative re-incision and drainage could be largely eliminated if deep and numerous incisions are made during the initial surgical procedure. A careful search for areas that may be involved is needed. This can be accomplished by the correlation of anatomic possibilities.

TABLE 7

*Complications*

| TYPE OF TREATMENT | NO. OF CASES | PER CENT |
|---|---|---|
| A. Re-incision and drainage | 26 | 17.3 |
| B. Urethral fistula | 21 | |
| | 7 healed | 33 |
| | 3 had plastic repair | |
| C. Hemorrhage | 7 | 4.7 |
| D. Pneumonia | 26 | 17.3 |

The second urethral fistulae may be present when the patient is first seen. If it does not heal on catheter drainage, plastic repair with excision of the surrounding scar can be carried out.

The third complication may occur in spite of careful hemostasis at the operating table, by progression of sloughing necrotic tissue through the course of blood vessels. Adequate control of bleeding with clamps and ligatures, in addition to replacement therapy must be promptly accomplished.

Most of the patients that died of pneumonia were in the prechemotherapy group.

## SUMMARY AND CONCLUSIONS

One hundred and fifty cases of urinary extravasation or, perhaps a more accurate term, *peri-urethral phlegmon*, occurring in the Baltimore City Hospitals from 1935–1951 have been presented (2, 4, 6, 10, 11). A review of the anatomic, etiologic, symptomatologic, and bacteriologic aspects of the condition are discussed. The treatment, operative and supportive is given. The mortality and morbidity are presented.

Perhaps in cases where no peri-vesical extension of phlegmon has occurred, external urethrotomy can replace suprapubic cystostomy for diverting the urinary stream. In extensive cases it appears that it would be preferable to do suprapubic drainage be cause of the difficulty of ascertaining any spread behind the urogenital diaphragm.

The effect of chemotherapy and anti-biotic drugs is striking, in reducing the incidence of severe urethral strictures following urethritis. And also the remarkable reduction of mortality and morbidity once peri-urethral phlegmon has occurred.

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# DISSECTING ANEURYSM OF THE AORTA AND THE SUPERIOR VENA CAVA SYNDROME*

S. EDWIN MULLER, M.D.

In June, 1953 Calenda and Uricchio (1), in reporting on the superior vena cava syndrome, included one case of obstruction of the superior vena cava by a dissecting aneurysm of the aorta. A review of the "world literature" by these authors disclosed that in only one other instance had such an entity been described. This case was reported by Baker (2) in 1940.

In September, 1937 a 69 year old man was admitted to the medical service at Mercy Hospital and was presented on regular medical ward rounds to Dr. Maurice C. Pincoffs. A diagnosis of this entity was made at that time and confirmed 18 months later at autopsy. Apparently this combination of symptoms is extremely rare. For this reason the following case is presented. The tabulation of data on 6 other cases of dissecting aneurysm of the aorta seen at autopsy in this hospital is presented in Tables I and II.

## REPORT OF CASE

A 69 year old white male, hospital #29227, was admitted to Mercy Hospital on September 27, 1937 complaining of shortness of breath and cyanosis of the face, neck and upper extremities.

*He gave the following history:* Five years before admission he was treated for high blood pressure and "heart trouble." One year before admission while sitting in a chair he suddenly developed a severe pain in the region of his heart radiating into the back and left shoulder. He stayed in bed one week and after that time noticed increasing shortness of breath and the appearance of cyanosis of the face and upper extremities with the gradual increase in size of the superficial veins of the chest and abdomen.

He had recurrent osteomyelitis of the left tibia following an injury in early life. The past history and family history are otherwise noncontributory.

*Physical Examination:* The temperature was 99 F., pulse rate 90, respiration 30, blood pressure 160 systolic over 80 diastolic (right arm) 180 systolic over 70 diastolic (left arm). The skin of the face, neck, chest, upper back and upper extremities was deeply cyanotic. Large tortuous veins, about 7 mm. in the chest wall anteriorly and posteriorly became markedly engorged on coughing. These veins filled more rapidly from above downward.

Moist rales were present in both lung bases. Examination of the heart showed an enormous heave over the precordium. A thrill was felt over the base. The point of maximal impulse was felt at the 6th interspace in the anterior axillary line. The retro manubrial dullness was 10 cm. in the 2nd interspace. A harsh systolic with a faint diastolic murmur was heard at the aortic area. A loud mitral systolic murmur transmitted to the left axilla was also heard. The liver was palpable about 6 cm. below the costal margin. The large tortuous veins continued down over the abdomen. The extremities showed no edema. The peripheral vessels were sclerotic. Dorsalis pedis palpations were present.

*Laboratory Findings:* The hemoglobin was 13.4 gm., 4,700,000 red blood cells, 8,200 white blood cells, with a normal differential. Blood urea 29, blood sugar 76. STS was negative. Teleo roentgenogram showed the following:

| | |
|---|---|
| Width of vessels | 8 C.M. |
| Right heart | 5½ C.M. |
| Left heart | 12½ C.M. |
| Transverse diameter | 18 C.M. |

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* From the Mercy Hospital, Department of Medicine, School of Medicine, University of Maryland.

TABLE I

| CASE | AGE | SEX | COLOR | EXERTION PRIOR TO ONSET | ADMISSION DIAGNOSIS | INTIMAL TEAR | FINAL DIAGNOSIS | RUPTURE INTO PERICARDIUM |
|---|---|---|---|---|---|---|---|---|
| 1 | 72 | F | W | None | Coronary occlusion | Yes | Dissecting aneurysm 2 cm. above aortic valve through 1st portion of aorta | Yes |
| 2 | 51 | M | W | None awakened from sleep | Acute pancreatitis | Yes | Dissecting aneurysm from left subclavicular artery to 5 cm. above renal vessels | No |
| 3 | 75 | M | W | None | Coronary occlusion | Yes | Dissecting aneurysm 2 cm. above aortic valve to renal vessels | Yes |
| 4 | 35 | F | C | None | Dissecting aneurysm of aorta | Yes | Dissecting aneurysm 1 cm. above aortic valve extending 5 cm. | Yes |
| 5 | 63 | M | W | None | Coronary occlusion, Peptic ulcer | Yes | Dissecting aneurysm 15 cm. above aortic valve to diaphragm. 2 saccular aneursyms present in this area. Fusiform aneurysm left popliteal artery. "Double barreled aorta." | Hemorrhage into retro-peritoneal space and into peritoneal cavity. |
| 6 | 72 | M | W | None | Cerebral thrombosis | Yes | Dissecting aneurysm ascending aorta | Yes |
| 7 | 69 | M | W | None | Dissecting aneurysm of the aorta, superior vena cava syndrome. | Yes | Dissecting aneurysm of the aorta from aortic cusps to coelic artery. Superior vena cava syndrome. "Double barreled aorta." | No |

TABLE II

| CASE | DEGREE OF ATHEROSCLEROSIS | TYPE OF PAIN | HISTORY OF HYPERTENSION | AORTIC DIASTOLIC MURMUR | EKG FINDINGS | LIVED AFTER ONSET OF SYMPTOMS |
|---|---|---|---|---|---|---|
| 1 | Marked | Persistent substernal. No radiation | Yes | No | Coronary insufficiency | 14 hours |
| 2 | Marked | Back pain radiating to epigastrum. Abdominal rigidity. | No | No | Not done. | 24 hours |
| 3 | Marked | Pain between shoulder blades | No | Not reported but evidently present. B.P. 110/0 | Left bundle branch block | 48 hours |
| 4 | None | Precordial. Radiating into back. Pain in episternal notch on flexion of neck. | Yes | Yes | ST segment elevation with upright T waves suggesting pericarditis | 7 days |
| 5 | Marked | Aching pain in lower back. | Yes | No | Coronary insufficiency | 2 years |
| 6 | Marked | Not described. Coma | Yes | No | Not done. | 1 day |
| 7 | Marked | Precordial pain radiating into back and left shoulder | Yes | Yes | AV nodal rhythm. Left ventricular strain | 2 years |

| | |
|---|---|
| Oblique diameter | $21\frac{3}{4}$ C.M. |
| Width of base | $14\frac{3}{4}$ C.M. |
| Width of chest | 29 C.M. |

Marked enlargement of heart and aorta.
There is a marked dilatation in region of right auricle.
Fluoroscopic examination
Lateral oblique chest—Marked enlargement of the descending aorta. Diffuse in character. Lungs clear. Trachea displaced to left.
(a) aortic aneurysm, descending aorta.
(b) left cardiac enlargement chiefly left ventricle.
EKG impression: 1. AV nodal rhythm; 2. Left ventricular strain.

A diagnosis of dissecting aneurysm of the aorta with pressure on the superior vena cava was made. The patient remained in the hospital for one month and was discharged unimproved.

His second admission was on January 9, 1939, complaining of persistent swelling of the forearms and hands and intermittent swelling of the lower extremities. The chest, face and upper extremities were deeply cyanotic. The tortuous veins over the chest and abdomen were dilated. The hands and arms were markedly edematous. The lower extremities showed slight edema only. Breathing was labored. Moist rales were heard throughout both lung fields. Blood pressure was 160 systolic over 80 diastolic (right arm) 180 systolic over 78 diastolic (left arm).

*Course in hospital:* There was no improvement or symptomatic treatment. The patient's condition grew steadily worse. Death occurred on the second hospital day.

*Pathologic Findings:** The body is that of a white, elderly male. There is a marked pitting edema of the upper extremities, of the upper chest wall and a slight puffiness of the soft tissues of the neck. There is definite dilatation of the superficial veins of the neck, chest, and abdominal wall (intercostal veins also dilated) and some edema of the lower extremities. Hypertrophic arthritic changes are present in the phalanges of the hands. The peripheral arteries are palpably sclerotic. The liver edge may be felt to be about 6 cm. below the costal margin. A midline incision was used to open the body.

*Heart and aorta:* About 50 cc. of clear fluid is removed from the pericardial cavity. The heart and aorta are taken out intact. The heart is quite enlarged, primarily the left ventricular area. However, the other component areas of the heart are also enlarged, but not to the same extent.

There is a definite aneurysm occupying the right and posterior portion of the ascending and the beginning of the transverse aorta. The aneurysm is adherent to the anterior wall of the right auricle, from which it can be peeled away. The right auricle is definitely enlarged. This large aneurysm, about 5 cm. in diameter, creates pressure by its anatomic position on the superior vena cava. The left ventricle, the aortic ring and the anterior surface of the aorta are cut in a single line. The vessels springing from the aorta are cut as high as possible. The left ventricular wall is tremendously hypertrophied. There is a moderate degree of dilatation. The aortic cusps show some arteriosclerotic changes and thickening. The ascending aorta (intimal surface) shows very little arteriosclerotic change and of itself does not appear to be aneurysmal in character. However, there is an opening about 2 cm. in diameter with edges well healed and smooth at the junction of the ascending and transverse aorta. The opening apparently has broken through the intima and media and has dissected the right lateral surface of the ascending aorta into 2 separate layers, the outer layer forming the huge dilated aneurysmal sac.

This dissecting aneurysm has spread backward to the aortic cusps, upward, separating the coats of the major vessels springing from the aorta, and down the descending and thoracic aorta just proximal to the coeliac artery. It forms a tube occupying about one half the lumen of the descending aorta and opens into the main aorta, as mentioned previously, just proximal to the coeliac artery. There is one small aperture; midway between the proximal opening of the aorta into the dissecting aneurysm and the distal opening of the dissecting aneurysm into the aorta; about two mm. in diameter, between the 2 vascular passages. 1. There is definite arteriosclerotic placque formation covering the intima. 2. This is located just distal to the proximal opening of the aorta into the dissecting aneurysm, i.e., in the transverse aorta. At the junction of the distal end of the transverse aorta and the descending aorta, a definite arteriosclerotic coarctation is evident. The dissecting aneurysm seems to short circuit this arteriosclerotic narrowing of the true lumen of the aorta. However, the pressure of the dissecting aneurysm may have caused the arteriosclerotic narrowing at this point.

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* Autopsy performed by: Dr. W. C. Merkel; Dr. E. T. Lisansky; Dr. F. J. Vollmer.

The distal opening of the dissecting aneurysm into the aorta also reveals well healed smooth edges. It may be estimated that the lumen of the dissecting aneurysm is only slightly less than the remaining lumen of the aorta. A very pronounced amount of arteriosclerotic placque formation is noted in the abdominal aorta.

*Microscopic Notes:* Heart Wall: There is a hypertrophy of the individual muscle fibers with considerable scarring.

Aortic Branches: Sclerosis confined to intima and media. Some of the vessels show a splitting of the wall with granulation tissue filling the previously open area. For the most part there is extreme arteriosclerosis present.

*Final Pathologic Diagnosis:* 1. Arteriosclerotic cardiovascular disease; 2. Dissecting aneurysm of the aorta. Pressure on superior vena cava; 3. Arteriosclerotic coarctation of the aorta; 4. Cardiac dilatation and hypertrophy with fibrinous and myocardial degeneration; 5. Pulmonary edema; 6. Pleural effusion; 7. Congestion of dependent viscera; 8. Marked edema of upper extremities, moderate edema of lower extremities; 9. Edema of chest wall; 10. Marked enlargement of superficial veins of chest and abdomen; and 11. Hypertrophic arthritis.

## PATHOGENESIS

In reviews by Bauersfield (4) and Feldman (8), opinions concerning pathogenesis of dissecting aneurysm are discussed. The etiology is not definitely established. The present theories are summarized by Blain (13) as follows: Idiopathic cystic medionecrosis as described by Erdheim (12). This change has not always been found (4). The intimal tear has been considered the primary site of dissection. Blain (13) cites 16 cases in which no tear was found in the intima (3). Another theory holds that the dissection is caused by a ruptured vasa vasorum in the presence of medial degeneration. It may be concluded that no single cause can be demonstrated as consistently producing the changes leading to a dissecting aneurysm (10). Bauersfield (4) states that atherosclerosis cannot be the sole cause because this change is least common in the ascending aorta where the intimal tears usually occur. He also states that the tear frequently occurs between atherosclerotic plaques and rarely through one. This may be assuming that the intimal tear is the point of entrance of the dissecting force. True coarctation or narrowing of the aorta are cited as predisposing factors to dissection of the aorta (4). Hamilton and Abbott (3) in a review of 200 cases of coarctation found that 33 (16½ per cent) died of dissecting aneurysm. In 141 cases of dissecting aneurysm reported by Schnitker (5), 45 (32 per cent) had true coarctation or narrowing of the aorta. Narrowing of the aorta may be difficult to evaluate because the formation of a dissecting aneurysm may cause apparent narrowing of the lumen which did not exist before the accident. Syphilitic aortitis was formerly considered an important predisposing factor. In 141 cases of dissecting aneurysm Schnitker (5) found only 5 cases of syphilis. Weiss (9) and Sailer (10) state that in syphilis the process tends to fuse the layers of the aorta thereby hindering dissection. Hypertension was formerly considered important in the cause of dissecting aneurysm. Schnitker (5), in a review of 560 cases, found 80 per cent to be hypertensive. In the 141 cases under 40, only 50 per cent were hypertensive. Oppenheim (11) reported that it takes a pressure of 2 to 3 thousand mm. of Hg to rupture the aorta in fresh cadavers.

The course of the dissection may be varied. It may continue along the vessel and rupture back into the lumen forming "the double barreled aorta" as in 2 cases in this report. It may dissect a variable distance and stop. It may rupture through the adventitia into the retroperitoneal spaces, pleural cavities or into the pericardial sac. The last group is most commonly seen and occurs in 80–90 per cent with sudden death (8). The latter course occurred in 5 of the 7 cases recorded in this report.

## SYMPTOMS

The symptoms of dissecting aneurysm depend upon the vessels involved in the dissection. It has been mistaken for coronary occlusion, syphilitic aortitis, cerebral vascular accident, mediastinal tumor, diaphragmatic hernia, pancreatitis, cholecystitis, kidney stone, ruptured peptic ulcer, embolic occlusions, mesenteric thrombosis and spinal cord disease (8). Bay (6) states "when severe chest pain, especially upper back pain accompanied by cerebral symptoms, a change in blood pressure in one extremity only, hematuria, transient paralysis of legs, widened aorta, or a suddenly developing aortic diastolic murmur, dissecting aneurysm should be suspected." Profound shock without electrocardiographic changes of coronary occlusion and without evidence of gastrointestinal bleeding should also suggest this condition. A pericardial friction rub was heard in one case tabulated and in one reported by Feldman (8). Bauersfield (4) quoting Levine, states that he often uses the absence of this finding to differentiate a dissecting aneurysm from a myocardial infarction.

An aortic diastolic murmur is a frequent and important diagnostic finding in dissecting aneurysm of the aorta (7). It is thought to be caused by sagging of the aortic commissural attachments as a result of the intimal tear (8). Schnitker (5) found an aortic diastolic murmur in 24 per cent of 141 cases.

## SUMMARY

Seven cases of dissecting aneurysm of the aorta are presented. A history of hypertension was found in 5 cases. Intimal tears were found in 7 cases. Marked arteriosclerosis was found in 6 cases. Rupture of the aneurysm into the pericardium occurred in 4 cases. Aortic insufficiency was present in 3 cases. The "double barreled aorta" was seen in 2 cases who lived approximately 2 years after the onset of symptoms suggesting dissection. One case developed the superior vena cava syndrome, a rare complication of dissecting aneurysm of the aorta and is reported as the third to be recorded.

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# CLINICO-PATHOLOGICAL CONFERENCE

## From the Case Histories, University Hospital, Baltimore

### *Clinical History*

In July, 1952, while in Frederick City Hospital for urinary retention, A. H., a white male of fifty-eight years, experienced persistent diarrhea. Associated with this was abdominal distention, "cramps", and anorexia, but no nausea or vomiting. The urinary difficulties (frequency, nocturia, straining, and finally retention) were relieved by catheterization. He was discharged after six days. In late August, ankle edema appeared. The diarrhea which first troubled him earlier in the summer continued. Bloody and sometimes "tarry" stools were passed. This was considered the result of hemorrhoids. The patient lost twenty pounds of body weight, and became increasingly weaker. There was shortness of breath or chest pain.

Prior to his current illness, the patient had had inflammatory rheumatism when seventeen years of age. This was described as an atypical migratory arthritis. Later in his hospital stay the patient disclosed the fact that he had gonorrhea when sixteen years of age.

When A. H. was admitted to this hospital, he presented evidence of recent weight loss. The skin was warm (oral temperature, 100.2°F.), dry, and pale. The veins of the neck and upper appendages were engorged. There was no jaundice or cyanosis. Although the patient appeared haggard, he was mentally alert and cooperative. In the retinal vessels, increased light streak was observed. The oral and nasal passages were free of significant changes. A thrill was palpated in the right supraclavicular fossa. The thorax was asthenic and hyper-resonant. By percussion, the heart was found to extend leftward to the anterior axillary line. Auscultation discovered in the mitral area a soughing first heart sound. A harsh, high pitched sound was heard in the neck. This sound was related to a systolic thrill. The bladder fundus extended four centimeters above the pubic symphysis. Tenderness was elicited by deep palpation in the hypogastric zone. There was a large hydrocele of the left testicle. Anorectal examination revealed external hemorrhoids and a boggy, enlarged prostate. Varicose veins and edema existed in the lower extremities.

The laboratory repeatedly reported positive serologic tests for syphilis. The reagin titre was four. A similar test performed with cerebrospinal fluid was negative. The urine contained few leucocytes, erythrocytes and albumin. Two electrocardiograms were done, but were not helpful in making the diagnosis.

The treatment of this patient included hypercaloric diet, vitamins and terramycin. The hydrocele was aspirated to obtain 500 cc. of turbid, pale yellow fluid. Three hundred cubic centimeters of urine were obtained by inserting a # 20 Foley catheter.

Six days after admission 500 cubic centimeters of blood were injected. Although an operation was planned, this was temporarily precluded by the supposed appearance of fluid in the left pleural sac. Thoracentesis, attempted twice, failed to obtain pleural fluid. Because of irregular cardiac rhythm, digitalis and mercurial diuretics were given. Four days later an abdominal operation was performed.

On the tenth postoperative day the wound was noticed to be necrotic and sloughing. The body temperature was normal. The following day, the patient was so feeble after a brief stay in a chair that he could not return to his bed. His blood pressure was 90/60 millimeters of mercury. He had at this time no pain or abdominal distention. Emesis discharged a large volume of green fluid. One hour later a meal was taken without complaint, but also without relish. Still another hour passed and then the patient experienced abdominal pains. He died thirty minutes later.

### *Clinical Discussion*

*Dr. Henry J. Marriott:* The abdominal distension preceding diarrhea suggests that there is partial obstruction, and the cramping pain suggests that the lesion is in the colon. If he noticed that his hemorrhoids got bigger at the same time that there was edema, it suggests that the edema was secondary to a venous congestive state. In other words, the edema was either on the basis of congestive failure or venous obstruction. Notice that we have venous congestion without dyspnea. Marked venous congestion in the neck suggests congestive failure, pericarditis or superior vena caval

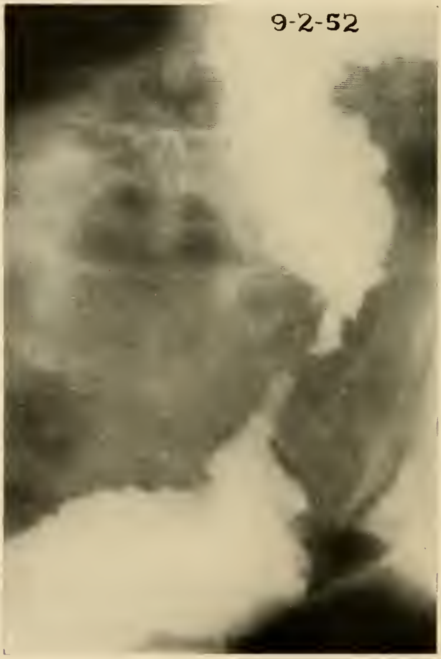

FIG. 1

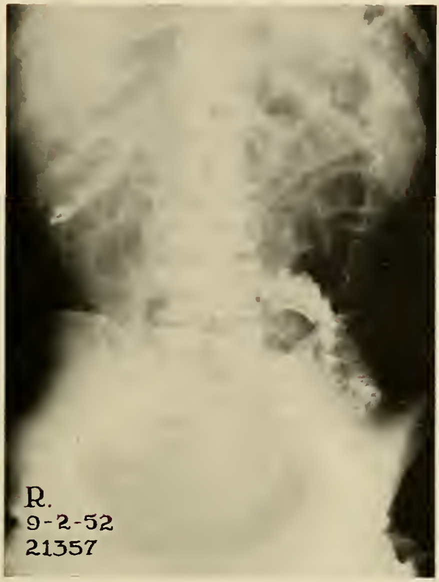

Fig. 2

syndrome. The murmurs plus the thrill suggest first of all aortic stenosis which might be on a rheumatic basis or sclerotic basis. They could be due to aortic aneurysm. Endocarditis involving the aortic valve is another possible cause of such a thrill.

*Dr. W. L. Kilby:* The x-ray examination of this patient's colon was done on September 2, 1952. Almost immediately after the barium enema was started evidence of a constrictive lesion was found in the mid-sigmoid. In this film, (Figs. 1 and 2) one sees the extension of the barium along the colon for about 4 cm. before the stream is markedly narrowed. Notice in the post evacuation film (Fig. 2.) the small filling defects in the mucosal pattern. Instead of having the fine feathered mucosal surface, you see that the surface is quite coarse, as if there were small pinpoint areas of rarefaction or filling defects. In other words, the mucosa of the colon has a hobnail pattern. This gives the impression that polyposis is present. However, this hobnail pattern could represent an inflammatory lesion. You notice that the defect is funnel-shape. Usually a malignant lesion begins abruptly and ends abruptly. Whether this is a malignant lesion or an inflammatory lesion is at present unanswered. A periocolonic abscess, pericolonic adhesions or a malignant lesion in close proximity to the colon could produce the findings seen in these films.

*Dr. Marriott:* Is that lesion in relation to the bladder?

*Dr. Kilby:* No, it isn't. Films of the patient's chest, taken September 3, represent a tortuous aorta. The first portion of the aorta is dilated. There is moderate cardiac hypertrophy with prominence of the left ventricle. Notice the calcium deposits in the aortic notch and also in the first portion of the aorta. Calcium in the first portion of the aorta is usually due to a luetic aortitis, but in only twenty per cent of cases of aortitis does calcification appear. On September 9, 1952, we saw fluid in the base of the left pleural cavity.

*Dr. Marriott:* This patient, while a boy of sixteen years, developed gonorrhea. At the age of seventeen he had "rheumatism." Two years ago, two months before admission, at the age of fifty-six, he developed urinary symptoms which culminated in complete obstruction and urinary retention. During the two months he lost a good deal of weight. At about the time the urinary retention began, he developed diarrhea, abdominal distension, and cramping pains in the lower abdomen. A week before admission he noticed edema of his ankles and an increase in the size of his hemorrhoids. He appeared to have heart failure, although he had not complained of dyspnea. He had albuminuria and hydrothorax. We have four main "pictures" here: the urinary, the cardiovascular, the alimentary or intestinal, and the sudden death. I have dealt briefly with the obstructive uropathy. Notice that there was no renal failure. His urea nitrogen was not above normal. The two probable causes of this are prostatism or urethral structure. Secondly, consider the cardiovascular lesions. I would have said that the cardiac lesion was aortic stenosis, but having seen the x-rays I think the dilatation would be enough to account for the murmurs and the thrill. The left ventricle does not look as large in the roentgenogram as it seemed clinically. Are we going to attribute the venous distension in the arms and legs all to congestive failure? I think we may, but on the other hand, the combination of tortuous distended vessels in the legs with quite marked edema makes one think of intra-abdominal thrombosis. Remember that thromboses are much more common with malignant disease than in any other cachectic condition. In considering the alimentary side of this patient's illness, the first thing that comes to mind is colonic carcinoma. This is particularly encouraged, I think, when one has abdominal distension which suggests a partial obstruction and cramps which localize in the colon. There are other colonic conditions to think of, one of which is diverticulitis. Also, polyposis without malignant degeneration comes to mind. The roentgenograms suggeted this diagnosis. One would think also of tuberculosis of the colon. Most colonic tuberculosis is secondary to a lesion somewhere else in the body, and we have no evidence of such in this patient. Other causes of diarrhea one might think of are gastrojejunocolic fistula and portal congestion. The low urea nitrogen and absence of liver enlargement moves against these possibilities. The suddenness with which death occurred indicates possibly pulmonary embolism, myocardial infarction or internal hemorrhage. None of these is considered likely because the symptoms presented, except for the precipitate occurrence of death, were not conclusive.

By overtaxing one's credulity the various phases of this man's illness may be converged in any of the following diagnoses: subacute bacterial endocarditis with mesenteric embolism; syphilis with aortic and intestinal lesions; primary amyloidosis; or carcinoma of the rectum. He probably had benign prostatic hypertrophy, chronic prostatitis and urethral stricture. Secondly, I believe that he had polyposis and carcinoma of the colon. Thirdly, I propose aortic dilation and finally, pulmonary embolism. All of these are based on probability and not on conviction.

*Senior Student:* What percentage of polyps would be seen by proctoscopy in polyposis of the colon?

*Dr. Hugh R. Spencer:* I should think seventy per cent.

*Senior Student:* Did you consider aortic aneurysm?

*Dr. Marriott:* No. Why do you consider it?

*Senior Student:* The terminal episode might be caused by rupture of the aneurysm.

*Dr. M. C. Pincoffs:* Dr. Spencer, did you estimate the duration of the peritonitis?

*Dr. Spencer:* I think it was of at least several days' duration. There was no organization found at autopsy.

### *Pathological Discussion*

*Dr. Hugh R. Spencer:* After death, the infected abdominal incision was opened to allow the escape of noisome, green-brown, purulent fluid from the abdominal cavity. A disrupted colotomy wound, found in the sigmoid colon, was considered the source of the peritoneal infection. Loops of ileum were adhered to the colon at this point. When the colon was opened, an estimated 250 sessile and pedunculated lesions were discovered distributed through the transverse, descending and sigmoid segments.

The heart was normal in size and conformation. The aortic valve was calcified. Luetic mesaortitis was evinced by corrugation of the intima and appropriate microscopic features.

Contrary to the clinical estimate, the prostate gland was small and of normal consistency. The bladder was dilated and distorted by diverticula. The largest diverticulum, arising in the apex, in capacity equaled the bladder. The ureters were dilated. The pelvic mucosa was hyperemic and edematous.

### *Anatomical Diagnosis*

Multiple polyposis, transverse colon, descending colon and sigmoid; recent colotomy; perforation of colon at suture line; generalized peritonitis with ileus; dilatation of stomach; diverticula, urinary bladder (basis of uretharal structure); cystitis; pyelitis; calcification of leaflets, aortic valve (old rheumatic); luetic mesaortitis.

# OBSTETRIC CASE REPORT*

The patient is a 39 year old para 0-0-4-0 whose last menstrual period was on the 21st of May, making the estimated date of confinement the 28th of February. A physical examination was essentially negative revealing a pregnancy of 6 to 8 weeks duration.

The past obstetric history reveals a spontaneous abortion of 20 weeks gestation in 1942. The following year a uterine suspension and appendectomy was performed. Three spontaneous abortions of 8 weeks duration followed in 1943, 1945, and 1947. The patient and her husband then had a complete infertility study. The only positive finding was a BMR of minus 29. She was placed on proloid gr. 1, 3 times daily and has continued that therapy for 6 years although her BMR was not followed.

Laboratory examination on the first of August revealed the BMR to be minus 15. On July 30 serum chorionic gonadotropin showed 64000 international units per 24 hours and urinary pregnandiol was 2.5 mgm per 24 hours. The patient was given 12.5 mgm of progesterone intramuscularly daily. This was continued since the urinary pregnandiol was 23.1 mgm. per 24 hours in August and 35.2 mgm per 24 hours in September. The serum chorionic gonadotropin showed 5000 I U per 24 hours, in September and in October it was 5620 I U per 24 hours which shows a fall below normal for this duration of pregnancy. Progesterone was then increased to 25 mgm daily.

The pregnancy was uneventful until the 36th week when the membranes ruptured spontaneously. On admission, the hospital examination revealed the height of the fundus to be 26 cm. ROA, and F. H. RLQ. The estimated size of the infant was 2500 gms. A rectal examination showed the cervix to be long and undilated with the vertex one centimeter above the spines. Because of the bad obstetric history, age of the patient, and premature rupture of the membranes a laparotrachelotomy under spinal anesthesia was performed resulting in a premature female infant weighing 2350 gms. The convalescence of the mother and baby was uneventful.

Comment: The incidence of spontaneous abortion in general, is very close to 10 per cent of all pregnancies. These can be divided into 2 categories. The first group are those that result from random factors that are not likely to repeat themselves, except by chance, and very little treatment is needed. On the other hand, there is a second group in which some condition impairs the growth of the ovum in each pregnancy. Bishop and others have arrived at an incidence of 0.41 per cent of all pregnancies. On the basis of this estimate the spontaneous cure rate for all abortions drops from 86.6 per cent after the first abortion to 16.4 per cent after the third habitual abortion. Hence it is important to determine the cause if possible.

This patient showed a low thyroid function which was treated with Proloid. In one study on habitual abortions this deficiency was noted in 72 per cent, and was the sole factor in 44 per cent of the cases studied.

Estrogen, progestrone and chorionic gonadatrophin are responsible for the maintenance of the decidual bed. Since a drop was noted on about the 130th day of pregnancy in this patient, there was allowance for a more rational indication for an increase in the dosage of progesterone.

The treatment, as outlined in this case, has met with a reported 67 per cent success in 43 pregnancies. However, it is not too unusual to see patients who have had 6 or 7 consecutive abortions and who, without treatment or other adequate explanation, go through a full term pregnancy without mishap.

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* From the Department of Obstetrics, University of Maryland School of Medicine, Baltimore.

# BOOK REVIEWS

**The Treatment of Injuries to the Nervous System.** *Donald Munro, M.D. W. B. Saunders Co., 1952, 284 pp., price $7.50.*

In this volume, written primarily for the general surgeon and general practitioner, are chapters concerning the treatment of injuries to the cranial nerves, spinal roots, peripheral and autonomic nervous system. The diagnosis, surgical treatment, and clinical management of injuries to the skull, brain, spinal cord, and cauda equina are very adequately, concisely and practically presented.

The author, a widely recognized authority on trauma to the nervous system, has long been interested in the rehabilitation of such patients. It is not surprising then to find also chapters entitled: "The Cost of Medical Care in Paraplegia and How It May Be Modified by Rehabilitation Services", "The Responsibility of the Hospital Trustees and Staff", and "Rehabilitation". Perhaps because it is a matter also which frequently comes to the attention of general surgeon and general practitioner, there is included a chapter entitled: "The Effect of Spinal Anesthesia on the Central Nervous System". The author states that these are the methods which have been evolved for and are in daily use in the treatment of all types of injury to the nervous system at the Boston City Hospital.

This reviewer, as well as several authors in the recent literature, would take exception to Dr. Munro's reluctance to have tracheotomy done in neurosurgical patients. In virtually every respect, however, his methods would receive universal endorsement. Particularly admirable is his work on the treatment of uncinate herniation. One should heartily recommend this volume to all who have to care for injuries to the nervous system. It will prove also an invaluable manual for the surgical house officer.

William H. Mosberg, Jr., M.D.

**The Human Pelvis.** *Carl C. Francis. With 61 illustrations, including 3 in color, C. V. Mosby Company, St. Louis, 1952. (191 pages of text, 18 pages of index).*

The material is divided into 12 chapters: osteology, ossification, joints, pelvic girdle, muscles and fasciae, vessels, nerves, pelvic intestine, urinary system, female genitalia, male genitalia, embryology. After each chapter the reader will find a list of the latest publications on pelvic anatomy, quoted conveniently with author's name, full title, place and year of publication. A concise yet complete account of the anatomy of the pelvis and perineum, with frequent reference to clinical application of the anatomy. Variations and anomalies, especially those which are of importance for the clinician, are pointed out. The book is recommended especially for the extensive account of the anatomy of the osseofibrous part of the pelvis, to which 84 of the 191 pages of text are devoted. The author had at his disposal the rich collection of osteological material at Western Reserve University. Many of the data are recorded in tabulated form and will be welcome as a means of convenient reference. The publishers deserve special praise for the ample illustration material and its beautifully clear reproduction.

Eduard Uhlenhuth, Ph.D.

**Textbook of Surgery.** *Edited by H. F. Moseley, M.A., D.M., M.Ch. (oxon), F.A.C.S., F.R.C.S. (Eng.) F.R.C.S. (c). Assistant Professor of Surgery, McGill University; Associate Surgeon, Royal Victoria Hospital, Montreal Canada. C. V. Mosby Co., St. Louis, 1952. 460 Text Illustrations, 46 Color Plates. 896 pages, $15.00.*

This text is outstanding because of its superbly chosen and excellent illustrations. Included are some 39 full page color illustrations by Frank H. Netter. The choice of line drawings and roentgenographs is excellent and their reproduction is admirable.

The book is written "to contain the principles of surgery and the amount of factual information required by students at the time of their graduation and during their early period as interns . . .". The authors are quite successful in attaining this goal. In several incidences the text is incomplete and may be misleading to the inexperienced.

Chapters included on Anesthesia, Plastic Surgery, the Abdominal Aspects of Gynecology, Amputations and the several chapters devoted to Orthopedic Problems make this a very valuable addition to the Surgical literature.

This *Textbook of Surgery* is highly recommended.

E. Roderick Shipley, M.D.

**The Rhesus Danger—Its Medical, Moral and Legal Aspect.** *R. N. C. McCurdy, London, England. William Heinemann—Medical Books, Ltd. pp. 138.*

The title, though a bit frightening, is one that has been given by the author to a book which deals with the problems that arise due to parental blood group incompatibilities. The seriousness of the many aspects are perhaps somewhat exaggerated because they are the author's own personal problems. Nevertheless, the book is an excellent contribution for both the layman faced with similar problems as well as the physician, lawyer or scientist associated with the legal, social and medical aspects of blood groups.

Part I is an excellent compact summary of the genetics, immunology, present status, and treatment of haemolytic disease as contributed by both American and British scientists. Part II deals with social problems that have arisen since the association of haemolytic disease with blood group incompatibilities. These problems include those of birth control, sterilization, abortion, artificial insemination and divorce. All of these have become social problems which merit greater study and perhaps modification since the discovery of the "purposeless effects" of blood group incompatibilities.

Elsa F. Jahn, B.S.

**Pharmacology in Clinical Practice.** *Harry Beckman, M.D. 839 pages 25 x 16.5 cm. W. B. Saunders, Co., Philadelphia, 1952, $12.50.*

This new book represents the experience gained in over twenty years of teaching pharmacology to medical and dental students at Marquette University and supersedes the Author's "Treatment in General Practice," which passes through six editions and enjoyed wide popularity for many years.

In general, the book is arranged along the same lines as its predecessor, but with much greater emphasis placed upon properties and uses of drugs than on clinical description of disease. The book is unique among pharmacology textbooks in that the historical development of drugs, the relationships of chemical composition and biological activity and mechanisms of drug action have been practically eliminated. In the reviewer's opinion, these omissions are unfortunate, for the approach to pharmacology from the viewpoint of disease entities rather than the conventional approach in which drugs are classified according to their chemical or pharmacologic properties will be a serious handicap to students with limited clinical experience. The book would serve as an excellent supplementary text in courses where there is close integration between pre-clinical and clinical subjects, and for advanced medical students.

The book is divided into two sections. Section I, presents the pharmacologic aspects of practically all major problems that arise in medicine and dentistry, grouped under nineteen headings (allergy, anesthesiology, autonomic pharmacology, cardiology, etc.). The disease or clinical condition is first described briefly in the Author's characteristic concise style. The use of drugs in the treatment of the disease or condition are then described in detail (dosage, side effects, absorption, excretion, etc.), usually accompanied by personal observations from the Author's experience. It is these personal observations as to the efficacy of certain drugs, expressed in Dr. Beckman's delightfully informal style that is one of the most attractive features of the book.

Section II, a compendium of drugs, represents only a small part of the book (53 pages). The drugs are listed in alphabetical order under proprietary, official and chemical names. In this section, physical and chemical properties, dosage forms and representative proprietary preparations are listed in encyclopedic style. This section of the book serves primarily for reference.

Dr. Beckman writes with great clarity and ease in an informal and engaging style. The book is beautifully printed and well bound. It is highly recommended to anyone interested in this fascinating subject, and would be especially useful to practitioners of medicine who are seeking information on recent developments in drug therapy. The book would be a valuable addition to any physician's library.

Raymond M. Burgison, Ph.D.

# MEDICAL SCHOOL SECTION

## PRESIDENT BYRD RESIGNS

Dr. H. C. Byrd resigned as President of the University of Maryland on January 1, 1954 and became a candidate for Governor of the State of Maryland. His resignation coincided with the beginning of the 1954 Primary Electoral Campaign.

# MERCY HOSPITAL NEWS SECTION

## VISITING STAFF NEWS

Dr. Frank K. Morris was recently appointed Chief of Gynecology, replacing Dr. Thomas K. Galvin, who died September 19, 1953.

Dr. Robert Z. Berry recently became a diplomate of the American Board of Otolaryngology.

Dr. Charles E. Brambel, Director of the Anticoagulant Clinic at Mercy, attended the Third Annual Symposium on Blood, Saturday, January 9, at Wayne University College of Medicine, Detroit.

Dr. C. Gardner Warner, Chief of Pathology, attended the National Convention of the College of American Pathologists in Chicago, October 12–16.

Recently appointed to the Mercy Hospital Visiting Staff are Dr. Margaret L. Sherrard Hamberry, Dr. Mary Matthews, and Dr. Michael Kevin Quinn.

Dr. Frederick J. Heldrich, Resident Pediatrician at Mercy 1951–52, has recently returned to Baltimore after serving in the Armed Forces. He has now taken up the practice of pediatrics at 11 E. Chase with Dr. Fred W. Smith.

Another returnee in the field of pediatrics is Dr. David Josephs, who will practice at 2161 E. Belvedere Ave. with Dr. Joseph Cordi.

## MEDICAL ALUMNI ASSOCIATION

The first annual Convention of the Mercy Hospital Medical Alumni Association will be held on Friday and Saturday, May 21 and 22, 1954. Plans have been formulated for a scientific and entertainment program under the Chairmanship of Dr. Daniel J. Pessagno. A complete program will be forthcoming in the next issue of the Bulletin.

## RESIDENT STAFF NEWS

Dr. August Kiel, Jr., is again back on the Mercy House Staff, as Resident in Neurosurgery, January 1 to June 30, 1954.

The following is a partial list of Resident and Assistant Resident appointments for the year 1954–55.

### *Surgery*

| | |
|---|---|
| Dr. Leonard G. Hamberry | Co-Resident in Surgery |
| Dr. William B. Rever, Jr. | Co-Resident in Surgery |
| Dr. Fausto M. Prezioso | Senior Asst. Resident Surgeon |
| Dr. Ira B. Anderson | Senior Asst. Resident Surgeon |
| Dr. Leonard H. Flax | Junior Asst. Resident Surgeon |
| Dr. Richard E. Schindler | Junior Asst. Resident Surgeon |
| Dr. George Schimert | Resident in Thoracic Surgery |

### *Medicine*

| | |
|---|---|
| Dr. William G. Esmond | Resident in Medicine |
| Dr. Robert E. Ensor | Senior Asst. Resident in Medicine (Oct. 15, 1954–Oct. 15, 1955) |
| Dr. Richard C. Fravel | Senior Asst. Resident in Medicine (July 1, 1954–Oct. 31, 1954) |
| Dr. Paul van Lith | Senior Asst. Resident in Medicine (August 1, 1954–July 31, 1955) |
| Dr. George H. Beck | Junior Asst. Resident in Medicine |
| Dr. Corbett L. Quinn | Junior Asst. Resident in Medicine |
| Dr. Harry S. Weeks | Second year General Practice Intern |

### *Obstetrics and Gynecology*

| | |
|---|---|
| Dr. Edward M. Barczak | Resident Gynecologist |
| Dr. Joseph T. Michels | Resident Obstetrician |
| Dr. Robert W. O'Conner | Asst. Resident in Gynecology and Obstetrics |
| Dr. George H. Miller | Asst. Resident in Gynecology and Obstetrics |

### *Otologyngology*

| | |
|---|---|
| Dr. Donald S. Carter | Resident Otolaryngologist |

### *Pathology*

| | |
|---|---|
| Dr. Jacento Gochoco | Resident Pathologist |

### *Dentistry*

| | |
|---|---|
| Dr. Nicholas C. Garcia | Resident Dentist |

### *Pediatrics*

| | |
|---|---|
| Dr. Liborio C. Garcia | Resident Pediatrician |
| Dr. Yng-Tsing Wong | Asst. Resident in Pediatrics |

## ITEMS

At a meeting of the Board of Governors, on January 14, 1954 it was decided to establish the Department of Pediatrics at Mercy Hospital as a separate department, independent of the Department of Medicine. This arrangement will be more in keeping with the School of Medicine and the University Hospital plan.

The Mercy Hospital has recently acquired from the City of Baltimore all the property from 311–331 St. Paul St., which for many years has been the site of the Department of Public Welfare, and of the Juvenile Court. This purchase means that the Sisters of Mercy now own the entire block, bounded by St. Paul Place, Pleasant, Saratoga and Calvert Streets. The deeds were turned over to the hospital on December 23, 1953.

The acquisition of this property is the first major step accomplished in Mercy's expansion program. Plans are already in effect for the raising of funds on a long range program, to meet the hospital's extreme need for additional facilities.

It is the expectation of the hospital authorities that, in the very near future, much progress will be made toward constructing a splendid structure—a modern hospital designed to meet urgent community needs.

## MEDICAL LIBRARY NOTES

The following donors presented gifts of books and journals to the library between November 1953 and February 1954:

Dr. Marie Andersch
Mrs. Ira E. Bayer, Jr.
Dr. Frank W. Hachtel
Dr. John C. Krantz, Jr.
Dr. Arthur M. Kraut
Dr. Morton M. Krieger
Dr. Carney Landis
Estate of Dr. Edward A. Looper
Mrs. Wilbur F. Skillman
Dr. John L. White
Dr. Huntington Williams

An anonymous contribution of $50 was added to the Ruth Lee Briscoe Library Fund in January. This might serve as an example of generosity for other alumni interested in building this library fund into a permanent endowment.

The professional schools' libraries of the University of Maryland were well represented in the October 1953 issue of the Bulletin of the Medical Library Association. It contained a professional article by Clara Louise Meckel, Librarian of the Psychiatric Institute; a book review by Simone C. Hurst, Librarian of the School of Nursing; and a guest editorial by Ida Marian Robinson, Librarian of the Schools of Medicine, Dentistry, and Pharmacy.

## NEW APPOINTMENTS TO FACULTY

1953–54

**Robert B. Dodd,** M.D., Professor of Anesthesiology and Head of Department. Effective Feb. 13, 1953.

**Lorin J. Mullins,** B.S., Ph.D., Visiting Professor of Psychiatry. Effective December 15, 1952 to February 15, 1953.

**John L. Peck,** M.D., Lecturer in Pediatrics. Effective February 1, 1953.

**Joseph M. White, III,** B.S., M.D., Lecturer in Pharmacology. Effective July 1, 1953.

**Elizabeth C. Heinz,** A.B., Junior Instructor in Bacteriology. Effective September 1, 1952.

**Joseph A. Jachimczyck,** M.D., Instructor in Legal Medicine. Effective July 1, 1953.

**Joseph H. Menning,** A.B., M.D., Instructor in Urology. Effective July 1, 1953.

**Kent E. Robinson,** M.D., Instructor in Psychiatry. Effective January 1, 1953.

**Jerome Styrt,** M.D., Instructor in Psychiatry. Effective January 1, 1953.

**Herbert L. Warres,** B.S., M.D., Instructor in Roentgenology. Effective July 1, 1952.

**James S. Brown,** M.D., Assistant in Anatomy. Effective February 1, 1953 to May 30, 1953.

**Donald B. Campbell,** M.D., Assistant in Obstetrics. Effective July 1, 1953.

**Joseph L. Doughton,** A.B., M.A., M.S.S., Assistant in Psychiatry. Effective September 22, 1952.

**William D. Gentry,** M.D., Assistant in Obstetrics. Effective July 29, 1953.

**James J. Gerlach,** A.B., M.D., Assistant in Otolaryngology. Effective July 1, 1953.

**Helen L. Guest,** A.B., M.S.S., Assistant in Psychiatric Social Work. Effective September 22, 1952.

**Leonard G. Hamberry,** A.B., M.D., Assistant in Surgery. Effective July 1, 1953.

**Stephen L. Magness,** A.B., M.D., Assistant in Medicine. Effective July 1, 1953.

**Arlie R. Mansberger, Jr.,** M.D., Assistant in Surgery. Effective July 1, 1953.

**Mary B. Matthews,** Assistant in Pediatrics. Effective July 1, 1953.

**William B. Rever, Jr.,** M.D., Assistant in Surgery. Effective July 1, 1953.

**Stanley R. Steinbach,** A.B., M.D., Assistant in Medicine. Effective July 1, 1953.

**John J. Tansey,** A.B., M.D., Assistant in Orthopaedic Surgery. Effective September 16, 1952.

**Ernest C. Young,** A.B., M.A., Assistant in Clinical Psychology. Effective October 1, 1952.

**Lester M. Libo,** A.M., Ph.D., Research Associate in Medical Psychology. Effective Jan. 29, 1953.

**Alice M. Band,** A.B., M.D., Baltimore Rh Typing Laboratory, Fellow in Medicine. Effective Jan. 1 to Jan. 31, 1953.

**Jane Wray Forrest,** Emerson Fellow in Pharmacology. Effective November 1, 1952.

**Luigi Frighi,** M.D., Fellow in Psychiatry. Effective September 1, 1952.

**Bella R. Hearst,** M.S., M.D., Research Fellow in Medicolegal Pathology. Effective Sept. 15, 1952.

**Ernest C. Herrmann, Jr.,** B.S., Fellow in Bacteriology. Effective September 1, 1952.

**Frank R. Holter,** M.D., Fellow in Psychiatry. Effective April 20, 1953.

**Paul W. Knowles,** John F. B. Weaver Fellow in Histology. Effective June 1 to Sept. 11, 1953.

**Norman W. Lavy,** A.B., Summer Fellow in Clinical Pathology. Effective June 1 to Aug. 31, 1953.

**Sze-Jui Liu,** M.D., Fellow in Medicine. Effective July 1, 1953.

**David H. Looff,** Fellow in Infectious Diseases. Effective June 1, to July 31, 1953.

**Fern E. MacAllister,** B.S., M.D., Fellow in Psychiatry. Effective April 15, 1953.

**Jack Mendelson,** Fellow in Psychiatry. Effective September 15, 1952.

**Giovanni Raccuglia,** M.D., Anna Corman Fellow in Hematology. Effective July 1, 1953.

**Mary V. Reed,** A.B., A.M., Bressler Reserve Fund Research Fellow in Bacteriology. Effective September 1, 1952.

**George Schimeri,** B.S., M.D., Fellow in Thoracic Surgery. Effective July 1, 1953.

**John O. Sharrett,** M.D., Fellow in Neurological Surgery. Effective July 1, 1953.

**Ursula T. Slager,** B.A., M.D., Hitchcock Fellow in Neuropathology. Effective July 1, 1953.

**Henry H. Startzman, Jr.,** M.D., Fellow in Roentgenology. Effective July 1, 1953.

**Andre J. Toussaint,** B.S., Fellow in Bacteriology. Effective June 1, 1953 to May 31, 1954.

**Emma Vestergaard,** M.D., Fellow in Psychiatry. Effective August 1, 1952 to June 30, 1953.

**Harry F. Wilson, Jr.,** B.S., Fellow in Pharmacology. Effective September 1, 1952.

**Dorothy Doe Adams,** A.B., Research Assistant in Psychiatry. Effective May 1 to June 30, 1953.

**Edna L. Barrabee,** Research Assistant in Psychiatry. Effective March 1, 1953.

**Frances S. Barbusca,** A.B., Research Assistant in Clinical Pathology. Effective June 1, 1953 to May 31, 1954.

**Elsa A. Burrows,** A.B., Research Assistant in Anatomy. Effective February 1 to Aug. 31, 1953.

**Florence M. Burnett,** B.S., M.S., R.N., Research Assistant in Psychiatry. Effective Sept. 1, 1952.

**Shirley A. Cox,** A.B., Research Assistant in Biochemistry. Effective July 1, 1953.

**Donald H. Dembo,** A.B., Research Assistant in Psychiatry. Effective June 15 to Sept. 15, 1953.

**David Freedenberg,** B.S., Bressler Reserve Fund Research Assistant in Pediatrics. Effective February 24, 1953.

**Marion C. Glass,** A.B., Research Assistant in Psychiatry. Effective September 22, 1952.

**Richard L. Glasser,** A.B., Research Assistant in Physiology. Effective June 1 to Aug. 31, 1953.

**Ruth K. Hall,** Research Assistant in Psychiatry. Effective Feb. 16, 1953.

**William M. Headley,** B.S., Research Assistant in Biochemistry. Effective June 1 to Aug. 31, 1953.

**Lydia D. Lipinski,** Research Assistant in Anatomy. Effective January 1 to August 31, 1953.

**John J. McLaren,** B.S., Research Assistant in Anatomy (Part-time). Effective October 1, 1952 to September 30, 1953.

**Eunice P. Kennelly,** Research Assistant in Psychiatry. Effective November 15, 1952 to June 30, 1953.

**June H. Mendelson,** B.S., Research Assistant in Psychiatry. Effective June 9, 1952.

**M. Joseph Rehak,** A.B., Research Assistant in Legal Medicine. Effective April 1, 1953.

**Martha K. Salter,** R.N., Research Assistant in Legal Medicine. Effective March 1, 1953.

**Arthur B. Silverstein,** A.B., A.M., Research Assistant in Psychiatry. Effective September 1, 1953 to August 31, 1954.

**Karl E. Sussman,** A.B., Research Assistant in Physiology. Effective June 16 to July 31, 1953.

**Barbara T. Stewart**, B.A., Research Assistant in Psychiatry. Effective November 15, 1952 to June 30, 1953.

**Herbert L. Yousem**, Research Assistant in Psychiatry. Effective June 18, 1953 to September 16, 1953.

**Betty May Zimmerman**, Research Assistant in Legal Medicine. Effective December 1, 1952.

## PROFESSORS EMERITI—1953-54

**Charles Bagley, Jr.**, M.A., M.D.—Professor of Neurological Surgery, Emeritus

**Waitman F. Zinn**, M.D.—Professor of Otolaryngology, Emeritus

**F. L. Jennings**, M.D.—Professor of Clinical Surgery, Emeritus

**Thomas R. Chambers**, A.B., M.D.—Associate Professor of Surgery, Emeritus

**Charles W. Maxson**, M.D.—Associate Professor of Surgery, Emeritus

## PROFESSORSHIPS BY PROMOTION

**William S. Love**, A.B., M.D. From Associate Professor of Medicine to Professor of Clinical Medicine.

## ASSOCIATE PROFESSORSHIPS BY PROMOTION

**Edward F. Cotter**, M.D. From Assistant Professor of Medicine to Associate Professor of Medicine.

**H. Vernon Langeluttig**, A.B., M.D. From Assistant Professor of Medicine to Associate Professor of Medicine.

**Ephraim T. Lisansky**, A.B., M.D. From Assistant Professor of Medicine to Associate Professor of Medicine and Associate in Psychiatry.

**Henry J. L. Marriott**, A.B., B.M. From Assistant Professor of Medicine to Associate Professor of Medicine.

**Samuel T. R. Revell, Jr.**, A.B., M.D. From Assistant Professor of Medicine to Associate Professor of Medicine.

## ASSISTANT PROFESSORSHIPS BY PROMOTION

**Robert E. Bauer**, A.B., M.D. From Associate in Medicine to Assistant Professor of Medicine.

**Edmund G. Beacham**, A.B., M.D. From Associate in Medicine to Assistant Professor of Medicine.

**Harry C. Bowie**, B.S., M.D. From Associate in Surgical Anatomy to Assistant Professor of Surgery.

**Charles F. Bramble**, A.M., Ph.D. From Associate in Medicine to Assistant Professor of Medicine.

**Henry C. Freimuth**, Ph.D. From Associate in Legal Medicine to Assistant Professor of Legal Medicine.

**John S. Haines**, A.B., M.D. From Associate in Urology to Assistant Professor of Urology.

**Meyer W. Jacobson**, A.B., M.D. From Associate in Medicine to Assistant Professor of Medicine.

**Samuel Legum,** M.D. From Associate in Medicine to Assistant Professor of Medicine.

**William V. Lovitt, Jr.,** B.S., M.D. From Associate in Legal Medicine to Assistant Professor of Legal Medicine.

**George G. Merrill,** M.D. From Associate in Neurology to Assistant Professor of Neurology.

**S. Edwin Muller,** A.B., M.D. From Associate in Medicine to Assistant Professor of Medicine.

**Robert T. Parker,** A.B., M.D. From Instructor in Medicine to Assistant Professor of Medicine.

**Lawrence M. Serra,** M.D. From Associate in Medicine to Assistant Professor of Medicine.

**Merrill J. Snyder,** B.S., Ph.D. From Instructor in Bacteriology to Assistant Professor of Medicine in Clinical Bacteriology.

**Raymond K. Thompson,** B.S., M.D. From Associate in Neurosurgery to Assistant Professor of Neurological Surgery.

## ASSOCIATES BY PROMOTION

**R. Adams Cowley,** M.D. From Assistant in Thoracic Surgery and Assistant Director Experimental Surgery to Associate in Surgery.

**William F. Cox, III,** M.D. From Instructor in Medicine to Associate in Medicine.

**Perry O. Futterman,** A.B., M.D. From Instructor in Medicine to Associate in Medicine.

**Donald W. Mintzer,** M.D. From Instructor in Medicine to Associate in Medicine.

**Clarence P. Scarborough,** M.D. From Instructor in Medicine to Associate in Medicine.

**Leonard Scherlis,** B.S., M.D. From Instructor in Medicine and Pediatrics to Associate in Medicine.

**Stephen J. Van Lill,** A.B., M.D. From Instructor in Medicine to Associate in Medicine.

## INSTRUCTORSHIPS BY PROMOTION

**Marjorie R. Fleitzer,** B.S., M.S.S. From Assistant in Psychiatric Social Work to Instructor in Psychiatric Social Work.

**Marvin Goldstein,** A.B., M.D. From Assistant in Medicine to Instructor in Medicine.

**Joseph C. Matchar,** A.B., M.D. From Assistant in Medicine to Instructor in Medicine.

**Thomas M. Stevenson, Jr.** From Assistant in Art as Applied to Medicine to Junior Instructor in Art as Applied to Medicine.

## SUPPLEMENTAL as of June 18, 1953

### *New Appointments*

**Alfred T. Nelson,** M.D., Clinical Professor of Anaesthesiology. Effective July 1, 1953.

**Edmund P. Coffay, Sr.,** B.S., M.D. Assistant in Medicine. Effective July 1, 1953.
**William A. Niermann,** M.D. Assistant in Pediatrics. Effective July 1, 1953.
**Vahram Aratoon,** M.D. Assistant in Ophthalmology. Effective July 1, 1953.
**William F. Peterson,** M.D. Assistant in Gynecology. Effective July 1, 1953.
**Albert M. Powell, Jr.,** M.D. Research Assistant in Pediatrics. Effective July 1, 1953 through October 31, 1953.
**P. Govinda Menon,** M.D. Fellow in Pediatrics. Effective July 1, 1953 through June 30, 1954.

## SUPPLEMENTAL as of August 3, 1953

### *New Appointments*

**John R. Reid,** Ph.D., Professor of Philosophy in Psychiatry. Effective July 1, 1953.
**Wm. G. Ebeling, III,** B.S., M.D. Associate in Medicine and Chief of the Division of Gastro-Enterology. Effective July 1, 1953.
**Wm. W. Magruder,** M.D. Instructor in Psychiatry. Effective July 1, 1953.
**Miriam S. Daly,** A.B., M.D. Assistant in Pediatrics. Part-time. Effective July 1, 1953.
**Sarah V. Huffer,** M.D. Assistant in Psychiatry. Effective July 1, 1953.
**John H. Young,** A.B., M.D. Assistant in Psychology. Effective July 1, 1953.
**Ira N. Tublin,** B.S. Summer Fellow in Medicine. Effective July 1, 1953 to August 25, 1953.
**William J. Elton.** Research Assistant in Psychiatry. Effective July 1, 1953.
**Martin K. Gorten,** M.D. Research Assistant in Pediatrics. Effective July 1, 1953.

### *Promotions*

**James G. Arnold, Jr.,** M.D. From Associate Professor of Neurological Surgery to Professor of Neurological Surgery and Acting Head of the Department.
**Samuel L. Fox,** Ph.D., B.S., M.D. From Associate in Otolaryngology and Instructor in Physiology to Associate Professor of Otolaryngology and Instructor in Physiology.
**Benjamin H. Isaacs,** M.D. From Associate in Otolaryngology to Assistant Professor of Otolaryngology.
**F. Ford Loker,** M.D. From Instructor in Surgery to Associate in Surgery.
**Joseph M. Miller,** M.D. From Lecturer in Surgery to Associate in Surgery.
**Robert C. Sheppard,** M.D. From Instructor in Surgery to Associate in Surgery.
**Roderick Shipley,** A.B., M.D. From Instructor in Surgery to Associate in Surgery.
**William T. Supic,** M.D. From Instructor in Proctology to Associate in Proctology.
**John H. Hirschfield,** M.D. From Assistant in Otolaryngology to Instructor in Otolaryngology.

## RESEARCH DOG PLAN OPERATES SMOOTHLY

### *Ordinance Supported by Maryland Society for Medical Research Continues to Operate Satisfactorily*

Dogs from the Baltimore City Animal Shelter have now been available to the medical schools for the last thirty months. During this period Johns Hopkins University School of Medicine has requisitioned over 2500 animals and the University of Maryland School of Medicine over 1500. Thus something over 4000 dogs which otherwise would have been uselessly put to death in the Shelter's gas chamber have made a substantial contribution towards medical research and education. The cost of these dogs to the medical schools, if purchased from a dealer, would have been over $25,000, a sum which instead has been released for expenditure on some other phase of medical research or education. Baltimoreans may well be proud of the fact that they have created such an enlightened system in their own midst.

Ever since its inception, the ordinance passed by the City Council on December 15, 1949 and signed by the Mayor on December 17, 1949 has worked smoothly and efficiently. This in large part is due to the splendid cooperation given by the Department of Sanitation under the direction of Mr. William P. Fannon, Commissioner, charged with the responsibility of operating the pound. Further the law has worked clearly to protect the pets owned by citizens of the city. Under existing arrangements all dogs picked up by the city are held at the animal shelter for at least seven days before being either killed or turned over to the medical schools. Actually the law defines the holding period as only four days but this was extended by personal order of Mayor D'Alesandro when the city took over operation of the pound in January 1950. Thus pet owners whose dogs are not licensed or are not wearing their license as the law requires, are insured an ample opportunity to reclaim their animals.

Actually fewer stray dogs are killed under the law than otherwise would be the case for a certain small percentage of the animals turned over to medical schools are not used in sacrifice experiments. On occasion such animals are placed in homes if such can be found. The Baltimore Animal Aid Association cooperates in this phase of the work.

AMERICAN MEDICAL
EDUCATION FOUNDATION
535 N. Dearborn St., Chicago 10, Ill.

# POST GRADUATE COMMITTEE SECTION

## POST GRADUATE COMMITTEE, SCHOOL OF MEDICINE

HOWARD M. BUBERT, M.D., *Chairman and Director*
Elizabeth Carroll, *Executive Secretary*
Post Graduate Office: Room 600
29 South Greene Street
Baltimore 1, Maryland

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## SECOND POST GRADUATE TELEVISION COURSE PRESENTED

Under the joint auspices of the Postgraduate Committee of the School of Medicine, the Maryland Academy of General Practice and the Baltimore City Medical Society a closed circuit telecast of medical and surgical clinics, was presented on the afternoon of Thursday, November 5, 1953.

Carried by closed circuit facilities of station WBAL-TV the clinical demonstration were seen simultaneously by the Academy of General Practice in the Gordon Wilson Hall of the University Hospital and by members of the Baltimore City Medical Society in the Osler Hall of the Medical and Chirurgical Faculty.

The cost of producing and distributing the program was made possible through a grant from the Read's Drug Stores.

It was as follows:

### CLOSED CIRCUIT TV PROGRAM

12:00 Noon Luncheon
12:45 p.m. Music
12:50 p.m. Introduction
**Howard M. Bubert,** M.D., Chairman and Director Postgraduate Committee
**H. Boyd Wylie,** M.D., Dean, School of Medicine University of Maryland
**Lauriston L. Keown,** M.D., President, Maryland Academy of General Practice
**Wetherbee Fort,** M.D., President, Baltimore City Medical Society
1:00 p.m. Gynecologic Procedure of Interest to the General Practitioner
**J. Mason Hundley, Jr.,** M.D., and Staff
1:30 p.m. Clinical Pathologic Conference
**Maurice C. Pincoffs,** M.D.
**Hugh R. Spencer,** M.D.
1:55 p.m. Cardiac Clinic
**William S. Love, Jr.,** M.D.
**Sidney Scherlis,** M.D.
**Leonard Scherlis,** M.D.
**C. Edward Leach,** M.D.
2:30 p.m. Problem of Aortic Aneurysm
**Henry T. Bahnson,** M.D., Associate Professor of Surgery, The Johns Hopkins University School of Medicine
2:55 p.m. So-called Pregnancy Tumors of the Mouth
**Myron S. Aisenberg,** D.D.S., Professor of Pathology, University of Maryland School of Dentistry

3:10 p.m. Five-minute Intermission—Music

3:15 p.m. Anticipatory Pediatrics
**J. Edmund Bradley**, M.D., and Staff

3:35 p.m. Atopic Eczema and the Allergic Dermatoses Affecting the Hands
**Harry M. Robinson, Sr.**, M.D.
**Harry M. Robinson, Jr.**, M.D.

4:05 p.m. The Rh Factor in Obstetrics—A Symposium
**Isadore A. Siegel**, M.D., Obstetric Aspects
**Martin K. Gorten**, M.D., Pediatric Aspects
**Milton S. Sacks**, M.D., Laboratory Interpretation

4:35 p.m. Operative Clinic—"Lumps in the Neck"
**Harry C. Hull**, M.D.

## THE MEDICAL SCHOOL AND HOSPITAL PLATES

Plates of the School of Medicine; the old Hospital and the new Hospital; University of Maryland, are available. These white plates are 10 inches in diameter with the design printed in black.
The price is $2.50 each, plus fifty cents insurance and postage in the U. S. A.
Insurance and postage for foreign mail is one dollar. Please send your order, with check, stating the plates desired to Mrs. Bessie M. Arnurius, Box 123, University Hospital, Baltimore 1, Maryland.
Checks should be made payable to the NURSES' ALUMNAE ASSOCIATION OF THE UNIVERSITY OF MARYLAND.

# ALUMNI ASSOCIATION SECTION

OFFICERS

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*Vice-Presidents*

John Mace, Jr., M.D.
John M. Beachley, M.D.
C. Wilbur Stewart, M.D.

Simon Brager, M.D., *Secretary*
Edwin H. Stewart, Jr., M.D., *Assistant Secretary*
Minette E. Scott, *Executive Secretary*
Thurston R. Adams, M.D., *Treasurer*

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William H. Triplett, M.D.
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Frank J. Geraghty, M.D.

*Nominating Committee*
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Arthur Siwinski, M.D.
Gibson J. Wells, M.D.
Emanuel Schimunek, M.D.

*Library Committee*
Milton S. Sacks, M.D.

*Alumni Council*
Louis H. Douglass, M.D.
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*Representatives to General Alumni Board*
Thurston R. Adams, M.D.
William H. Triplett, M.D.
John A. Wagner, M.D.

*Representatives, Editorial Board Bulletin*
Harry C. Hull, M.D.
Albert E. Goldstein, M.D.
Daniel J. Pessagno, M.D.

The names listed above are officers for the term beginning July 1, 1953 and ending June 30, 1954.

## PRESIDENT'S LETTER

First may I express my gratitude to all of you for the honor which you have conferred in selecting me as your President. I assure you I will try to prove myself worthy. Now may I appeal to you for support of your Alumni Association and at the same time of the School itself. In 1957 we celebrate the 150th Anniversary of the University of Maryland. We are already planning for a large celebration and will call on you for help in the not too distant future.

In the meantime may I extend to you a most cordial invitation to contribute to the Bulletin, which is, after all, your Journal. Papers on various medical subjects from any member of the Alumni Association will be considered and, if acceptable, will be published. Another suggestion which comes to mind is that many of us have interesting cases of one kind or another. Why not send these in to the Editor as case reports and either write a discussion yourself or request that one be written by an authority on the subject? In the latter event you yourself would benefit by this comment and many of your colleagues would increase their knowledge. The case presented need not be extremely rare but should contain some interesting aspects of the disease.

I am constantly amazed at the continually increasing amount of instruction given in our medical schools. It appears that something is always being added but little is ever deleted. The medical student of today has to do a great deal more work than he did a few years ago. This means that he must be better prepared; have a much wider field of knowledge and must have acquired the ability to accept and retain a great deal more information. His study habits have to be well developed. He should have a willingness to subjugate all his interests to medicine. Should any of you know youngsters who are interested in studying medicine You as physicians and surgeons will do an immense service to prospective medical students saving them a great disappointment, by advising them either before or during their premedical courses. Emphasize the need for close application and the necessity of high scholastic standing required to be accepted by a medical school. The competition is stiff, the need for physicians is great. No school can afford to accept students who cannot meet normal competition or who cannot pass State Board examinations. As a member of the Committee on Admissions to our own School it has been my unpleasant duty to have to vote to reject several applications. Some of these have been submitted by extremely nice, likeable chaps who had very poor records in their premedical courses, very probably on the basis of lack of application. They had failed to acquire the habit of concentration. The committee felt that they could not have made the grade in medical school. Probably many of these youngsters could have done well if only someone had talked to them earlier and impressed upon them the importance of continuous effort.

Louis H. Douglas, M.D.

## DR. FRED W. RANKIN TO RECEIVE 1954 ALUMNI HONOR AWARD

Dr. Fred W. Rankin, one of the nation's outstanding surgeons and a member of the class of 1909, has been named the recipient of the 1954 Alumni Honor Award and gold key. He will be present at the Alumni reunion on June 3, 1954 and will deliver the principal address.

Dr. Rankin was born on December 20, 1886 in Mooresville, North Carolina, the son of Watson and Margaret (Houston) Rankin. In 1905 he graduated from Davidson College and 4 years later received the degree of Doctor of Medicine at the University of Maryland. Immediately following his graduation he spent 3 years at the University Hospital in Baltimore in the department of surgery, serving under Dr. Frank Martin. On July 1, 1912, he entered into private practice in Baltimore and continued his association with his Alma Mater with the position of Instructor in Surgery.

In 1916 he was appointed Assistant Surgeon at the St. Mary's Hospital, Mayo Clinic at Rochester, Minnesota, serving from 1916–1922. In 1922 he became Professor of Surgery at the University of Minnesota Medical School and the Mayo Foundation. In 1933 he returned to Kentucky and became surgeon at St. Joseph's Hospital at Lexington. Since 1941 he has been Clinical Professor of Surgery at the University of Louisville.

In World War I, Dr. Rankin served for 17 months in the Medical Corps with the rank of Major and was attached to the 1st A.C., 4th and 26th Divisions in France. He was Commanding Officer of Base Hospital #26. Upon his return to America he became Colonel in the Medical Reserve Corps, United States Army.

In World War II, Dr. Rankin's abilities were recognized by the Surgeon General. He was appointed Chief Consultant in Surgery as Assistant Surgeon General, with the rank of Brigadier General in the Army of the United States.

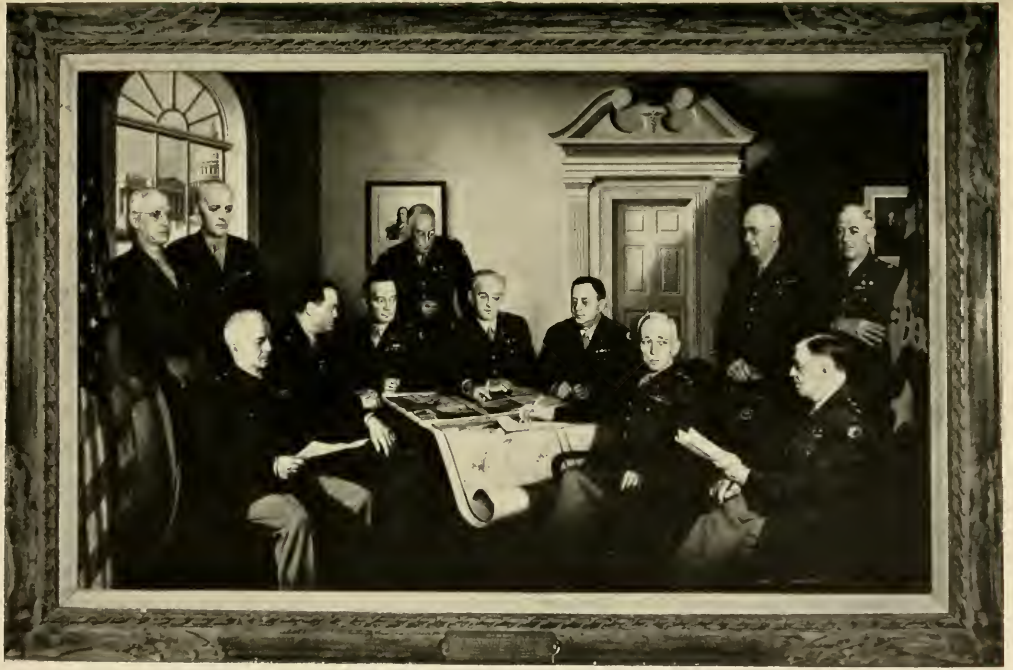
For his services he was awarded the Distinguished Service Medal, Victory Ribbon,

Dr. Fred W. Rankin

E.T.O. Ribbon, Asiatic Pacific Ribbon, American Theatre Ribbon and the Cross of Chevalier of the Legion of Honor (France).

Dr. Rankin has been a Fellow of the American College of Surgeons since 1920 and is currently President (1953–54). He is also a member of the American Medical Association, (President, 1942), serving on many committees, (Medical Preparedness, 1940–42), (Council on Medical Education and Hospitals, 1936–41), and (Chairman, Surgical Section, 1932–33). He is a member of the Interstate Post-Graduate Assembly (President, 1943), the American Surgical Association (President, 1949), the American Board of Surgery (Vice-Chairman), the Southern Surgical Association (Council Member and Past President) and the American Proctological Society. He is the author of over 300 scientific papers concerning principally abdominal surgery and surgery of the colon. He is the author of a widely read monograph "Surgery of the Colon" and is co-author of an additional monograph entitled "The Colon, Rectum and Anus".

Dr. Rankin holds honorary degrees from St. John's College, Annapolis, Maryland,



Reproduction of oil painting in Army Postgraduate Medical School, showing General Norman T. Kirk and Staff as it existed during World War II. Dr. Fred T. Rankin is fifth from the left. Left to right, the picture includes: Brig. Gen. Charles C. Hillman, U.S.A.; Brig. Gen. Hugh J. Morgan, U.S.A.; Brig. Gen. Raymond W. Bliss, U.S.A.; Col. James R. Hudnall, M.C.; Brig. Gen. Fred W. Rankin, U.S.A.; Lt. Col. Robert J. Carpenter, M.C.; Col. Edward Reynolds, M.A.C.; Brig. Gen. Raymond A. Kelser, U.S.A.; Maj. Gen. Norman T. Kirk, the Surgeon-General; Maj. Gen. Robert H. Mills, U.S.A.; Maj. Gen. George F. Lull, U.S.A.; Brig. Gen. James S. Simmons, U.S.A.

(M.A., 1915); Davidson College, Davidson, North Carolina, (Sc.D., 1937); University of Maryland, (Sc.D., 1939); University of Kentucky, Lexington, Kentucky, (Sc.D., 1942); Northwestern University, Chicago, Illinois, (Sc.D., 1943); University of Louisville, Louisville, Kentucky, (Sc.D, 1947) and Temple University, Philadelphia, Pennsylvania, (LL.D., 1943).

He has been invited to visit England during the Spring of 1954 to receive an honorary degree from the University of Leeds.

Dr. Rankin is a member of Beta Theta Pi, Phi Chi Medical Fraternity, Alpha Omega Alpha Honorary Fraternity, Phi Beta Kappa, and the Society of Sigma XI.

A distinguished alumnus, scientist and surgeon, Dr. Rankin has been selected for this high honor by unanimous vote of the Medical Alumni Association.

## CORRESPONDENCE

Dear Doctor Triplett:

Your letter of March 16 received and do appreciate the invitation to attend the 1904 class reunion. (JUNE 3, 1954 ED.). In 1948 or 1949 I did attend an Alumni reunion of the University not confined to the medical, and unfortunately failed to meet any of my class mates.

I really belong to the class of 1903, I had to leave college that year about 3 months before graduation, had the date recorded and returned the following year to complete the term and graduate.

In the mean time, I took the Arkansas State Board and began the practice of medicine the latter part of February 1903. I continued in practice there until date to return to finish my course, after which I returned to Arkansas. In 1905 I developed tuberculosis and went to Old Mexico January 6, 1906 and spent 26 years in Mexico except for the time spent as a 1st Lieutenant in the United States Army during World War I. I came to El Paso, Texas in 1931 and fortunately am still in active practice.

If you or any of the Alumni are in or passing through El Paso, please look me up.

Very sincerely
CARL E. JUMPER, CLASS OF 1903

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The following has been received from Dr. Dana W. Collison, P & S class of 1886.

January 31, 1954

Dr. William H. Triplett
Medical Alumni Association
Baltimore, Maryland

Dear Doctor:

Read your letter and invitation but at this time can not say as to accepting it but will let you know later. I would like to meet with you (at the Alumni Day exercises and class reunions on June 3, 1954—Ed.) and may be able to do so when the weather warms up at that time. Will let you know later on.

Dr. Dana W. Collison
779 Dennison Ave.
Columbus, O.
(P & S 1886)

## SCHOOL PHOTOS NEEDED

### *Alumni Requested to Search Files and Old Albums for U. of M. Pictures*

In the months to come the Bulletin is planning a photographic survey of the University of Maryland as it was and as it is, including photographs of professors, buildings now no longer in existence and some of the new additions to the School of Medicine. If you, as an Alumnus, have photographs in your possession which might contribute to this project, the Bulletin would be very grateful for their loan (or their gift), all prints to be returned if desired.

In submitting these pictures please give an appropriate legend on the reverse side or on a small slip of paper attached to the photograph. Consider personalities and buildings of utmost importance. Please think of the value of the photograph or reproduction in making the selection of the ones you plan to loan us. These pictures when printed will become a permanent record of the School of Medicine and no doubt will be of great historic value. All photographs should be mailed to the Bulletin of the School of Medicine, University of Maryland, Lombard and Greene Streets, Baltimore 1 Maryland.

## MARYLAND ACADEMY OF GENERAL PRACTICE ELECTS U. OF M. ALUMNI

Dr. Lauriston L. Keown, class of 1933, of Baltimore, Maryland was recently elected President of the Maryland Academy of General Practice. Dr. William T. Layman, class of 1939, of Hagerstown, Maryland was elected Secretary and Treasurer. Vice-Presidents include Drs. Nathan E. Needle, class of 1930 and Norman E. Sartorius, Jr., class of 1937, of Pocomoke City, Maryland. Dr. Robert W. Farr, class of 1934, was elected Director of the Eastern Shore and Dr. Hugh W. Ward, class of 1929 Director of Western Maryland. Delegates include Dr. Charles F. O'Donnell, class of 1944 of Towson and Dr. James Frankil, class of 1937 of Baltimore.

## ALUMNUS PROMINENT IN FRATERNAL ACTIVITY

### *Dr. Samuel S. Glick Serves as Phi Delta Epsilon Grand Counsul*

Dr. Samuel S. Glick of the class of 1925 recently was honored by the Phi Delta Epsilon Fraternity in being named Grand Counsul for the year 1953. An active practicing pediatrician, Dr. Glick served as an intern at the University Hospital from 1925 to 1926. He was then successively assistant resident at Sydenham Hospital and resident in pediatrics at the St. Vincent's Infant Home from 1927 to 1928. Until 1941 he served as Health Officer in the Baltimore City Health Department. In 1948 he was named assistant professor of pediatrics at the School of Medicine. He is a

Dr. Samuel Shipley Glick

pediatric staff member at the University Hospital, Sinai Hospital and the Lutheran Hospital of Maryland. Dr. Glick is a Fellow of the American Academy of Pediatrics, a member of the Baltimore City Medical Society, the American State Medical Society and a Fellow of the American Medical Association.

Dr. Glick's activity in the Phi Delta Epsilon Fraternity began during his student days. He was District Counsul in 1937.

## MANUSCRIPTS SOLICITED

Alumni of the School of Medicine have been urged by the Alumni Association to participate more actively in the Scientific Section of the Bulletin. The Editorial Staff of the Bulletin often receives items of general interest to the Alumni but in the past the majority of scientific papers have been published by members of the active staff of the School of Medicine. Alumni are reminded that the Bulletin welcomes contributions of a scientific nature. A prompt review and reply are guaranteed.

## THINK OF THIS

Do you know that from a total of $2,819,901.00 contributed to the American Medical Education Foundation, the School of Medicine, University of Maryland received as of September 30, 1952 a total of $37,498.00. The entire amount has been used to support the educational activities of the School of Medicine. Have you contributed?

## CLASS OF 1934 NOTE

*Class Plans Reunion June 3, 1954*

Dr. Leon H. Feldman, Flat Iron Building, Asheville, North Carolina, class of 1934 has expressed an interest in a twenty year reunion. The office of the Medical Alumni Association will gladly give the class of 1934 all necessary assistance in developing Dr. Feldman's desire into a reality this June.

## ITEMS

**Dr. Katherine V. Kemp,** class of 1948, has announced the removal of her office to 722 Stamford Road, Baltimore 29, Maryland.

**Dr. Raymond C. Vail Robinson,** class of 1940, has announced the opening of an additional office in the Northwood Professional Building, 1532 Havenwood Road in Baltimore.

**Dr. Raymond L. Markley,** class of 1946, has announced the opening of his office for the practice of gynecology at the Medical Arts Building in Baltimore.

**Dr. Milton A. Katz,** class of 1938, is active in general practice. His office is located at 1868 Pacific Avenue, Long Beach, California.

Dr. Milton A. Katz

**Dr. Jacob H. Conn,** class of 1929, has been elected a Fellow of the International Society for Clinical and Experimental Hypnosis. His article on "The Relation between Fatigue and Personality Factors in Severe Poliomyelitis" was published in a recent number of the A.M.A. Archives of Neurology and Psychiatry.

**Dr. Harold H. Weinberg,** class of 1945, has announced his return from active duty with the United States Army Medical Corps and the resumption of his practice which is limited to ear, nose and throat. Dr. Weinberg is now located at 1724 Eutaw Place and at 1413 Reisterstown Road in Baltimore.

**Dr. Norman Levin,** class of 1947, has announced his return from the armed forces and the opening of his offices for the practice of obstetrics and gynecology at 114 Medical Arts Building and 7013 Liberty Road in Baltimore.

**Dr. Leo F. Steindler,** class of 1908, has retired from service in the Veterans Administration and is now living in San Antonio, Texas. His address is 102 Suffolk Place, Zone 1.

**Dr. George F. Schmitt,** class of 1935, has recently been appointed Assistant Clinical Professor of Pharmacology at the University of Miami School of Medicine, Miami, Florida.

**Dr. Paul B. Toms,** class of 1945, is now located in Martinsville, Virginia where he is engaged in the practice of general surgery.

**Dr. Charles Shaw,** class of 1944, has recently returned from duty with the United States Navy and has opened his office for the general practice of medicine at 5801 Loch Raven Boulevard in Baltimore.

## MEMBERS OF 50 YEAR CLASSES, 1954

### UNIVERSITY OF MARYLAND, 1904

Mayer W. Aaronson
Charles Bagley, Jr.
Thomas G. Basnight
James R. Bishop, Jr.
S. Bernice Buck
Julian G. Busby
Harry F. Byrnes
William C. Chowning
Joseph Coleman
Alpheus E. Dann
Edgar B. Davis
Luis de Quevedo Garcia
Francis H. Digges
John Raymond Downes
Howard V. Dutrow
Arthur B. Eagle
Harlan L. Erwin
Arthur E. Ewens
William L. Funkhouser
Richard W. Garnett
William N. Gassaway
Jacob W. Gerber
Walton H. Hopkins
Robert E. Houston
James E. Hurley
Solomon C. Katzoff
John R. Lowery
Clyde C. Mack
Charles C. Orr
Charles L. Owens
Charles L. Parks
J. Oliver Purvis
Norman E. Sartorius
Patrick H. Tawes
Walter Van Sweringen
Henry Waldschmidt
William C. Webb
Myer A. Weinberg
Carson A. Willis
Silas G. Wright

## BALTIMORE MEDICAL COLLEGE, 1904

George C. Bicknell
Emmet R. Bucklew
Daniel E. Egan
Charles J. Hanson
Hermon E. Hasseltine
Carl E. Jumper
Carl F. Miller
William A. Nelson
Emil Novak
John Henry Orff
Lake Polan
Edmund A. Rogers
Howard G. Stevens
Fred B. Stuart
Matthew J. Sullivan

## COLLEGE OF PHYSICIANS AND SURGEONS, 1904

Morris D. Cohen
Edward M. Dailey
Joseph M. Ganey
Andrew C. Gillis
Perry B. Goodwin
Justin E. Hayes
Elisha S. Lewis
Edward F. McGinty
Alexander J. McNeil
Frank V. McVay
Wilber E. MacDougal
David C. Mock
John J. O'Donnell
John L. Opfermann
Leartus J. Owen
Clayton T. Ralls
Edwin R. Raymeley
Horace G. Ripley
Sidney M. Saltz
Reuben E. Schall
George H. Seaks
Joseph A. Trainor
James E. Wilson

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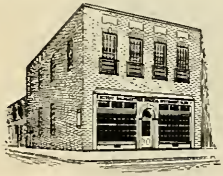

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# OBITUARIES

## Thomas Conrad Wolff, B.A., M.D., M.C.

After a prolonged and trying illness, Dr. Wolff died peacefully in his sleep at his summer home in Sturgeon Point, Ontario, at the age of 62.

Born in Canada on April 24, 1891, Dr. Wolff was graduated from McGill University in 1917 and then served two years overseas in the first world war as a Captain in the Canadian Army Medical Corps. From 1922 to 1927 he worked as a general practitioner in New England and then came to Baltimore for a year's postgraduate work at the Johns Hopkins Hospital. During this year he was persuaded to remain in Baltimore and he began practice here in the fall of 1928. He has been on both the Faculty of the University of Maryland School of Medicine and the Faculty of the Johns Hopkins School of Medicine almost since his arrival. He was soon put in charge of the teaching of Physical Diagnosis at the University, and for the past twenty years he has ably directed this important department. He was appointed an Associate Professor of Medicine in 1948.

Dr. Thomas Conrad Wolff (*Photo—Udel*)

For many years he has also served on the Medical Board of the State Industrial Commission and since 1937 he has been Medical Director of the Home for Incurables. Since 1930 he has been on the staff of Mercy Hospital as consultant cardiologist in charge of electrocardiography.

Dr. Wolff will long be remembered by many generations of Maryland graduates, especially as their kindly indoctrinator into the gentle art of physical diagnosis. He took great pride in teaching—which he thoroughly enjoyed—and it therefore goes without saying that he was a popular and excellent teacher. Among his many virtues, he was friendly, considerate and cheerful, and he will be missed by his colleagues and his student protégés alike. He is survived by his wife, two sons and a daughter. Too early in life he has been called out of a devoted family circle whose unusually happy atmosphere was a privilege for the visitor to enter.

## Dr. John F. Shea

Dr. John F. Shea, P & S, class of 1911, passed away in the Bridgeport (Connecticut) Hospital on June 12, 1953 at the age of 67. He was born in Holyoke, Massachusetts and attended the schools of his native city. He then entered the Williston Academy at Easthampton, Massachusetts where he was graduated in 1906. In the fall of 1907, Dr. Shea entered the College of Physicians and Surgeons at Baltimore. From the time of his enrollment until his graduation his scholastic ability was most brilliant, having led his class for all 4 years. At the graduation exercises he was class valedictorian.

After graduation he was appointed an intern at Bridgeport Hospital and continued there until he completed his residency. He then entered the practice of surgery in Bridgeport, Conn. which was interrupted to serve in World War I. On the termination of military service Dr. Shea resumed practice with the appointment of attending surgeon at the Bridgeport Hospital, a position he held until his death.

He married Miss Agnes Thompson of St. Mary's County Maryland, who survives him. He is also survived by a daughter, Mrs. Richard Shinn.

Dr. Shea was widely known throughout Connecticut as one of its outstanding surgeons. He was a member of the Founder's Group, American Board of Surgery, the Bridgeport Medical Society, the Connecticut Medical Society, the American Medical Association and the American College of Surgeons.

John F. Hogan, M.D.
P & S 1911

## Dr. Thomas K. Galvin

Dr. Thomas Keough Galvin, former Clinical Professor of Gynecology at the University of Maryland School of Medicine, died suddenly on Saturday, September 19, 1953, at the age of 61. A graduate of the College of Physicians and Surgeons where he received his medical degree in 1915, Dr. Galvin was for many years associated with the Gynecological Department of the School of Medicine. In addition, he served as Vice-President of the University of Maryland Medical Alumni Association and at the time of his death was a member of the Board of Directors.

Dr. Galvin joined the University of Maryland Army Hospital Unit early in the First World War, serving in France at Bazoilles-sur-Meuse. Upon his return to Baltimore in 1919, Dr. Galvin took up the practice of Gynecology and Obstetrics.

For many years he was a member of the Executive Committees of both St. Joseph's Hospital and the Hospital for the Women of Maryland and the Board of Governors at the Mercy Hospital in Baltimore. Dr. Galvin was Chief of Gynecology at both St. Joseph's and Mercy Hospitals.

A man with varied interests and abundant energy, Dr. Galvin served during the mid-thirties as the Chairman of the Building Committee for the Women's Hospital. During the Second World War, Governor Herbert R. O'Conor appointed him medical representative of the Appeals Draft Board.

In addition, Dr. Galvin served as Vice-President of the Baltimore City Medical Society. He was a former President of the Baltimore Obstetrical and Gynecological Society, a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons.

Dr. Galvin will be remembered as a man of many charities, a firm but friendly participant in community affairs and a credit to the profession of medicine.

FRANK K. MORRIS, M.D.

Dr. Thomas K. Galvin

## Dr. Ralph C. Achorn

Dr. Ralph C. Achorn, class of 1907, died suddenly at his late residence in Swampscott, Massachusetts, April 24, 1953. Dr. Achorn was born in Morrill, Maine, and received his medical degree from the School of Medicine in 1907. A man of wide interests, Dr. Achorn was a member of the Masonic Order, the Odd Fellows, numerous Church Societies, the Natural History Committee of the Appalachian Mountain Club, The Horticultural Society of Boston, the Brookline Bird Club and was a charter member of the Echology Society of Boston. He was also past president of the Boston Field and Forest Club, and an honorary member of the Forestry Department of New Hampshire. Dr. Achorn was recognized as an authority on trees and plants.

## Obituaries

**Achorn, Ralph Chase,** Swampscott, Mass.; B.M.C., class of 1908; aged 73; died, April 24, 1953, of coronary occlusion.

**Bell, Carl Wendell,** Raleigh, N. C.; P & S, class of 1914; aged 65; served during World War I; died, June 29, 1953, of coronary occlusion.

**Brown, Homer Scott,** Sutton, W. Va.; P & S, class of 1908; aged 72; died, September 24, 1953, of a heart attack.

**Brown, Joseph C.,** Lookout, W. Va.; B.M.C., class of 1895; aged 81; died, July 13, 1953, of arteriosclerosis.

**Bunnell, Frederick Norman,** Barnegat, N. J.; P & S, class of 1905; served during World War I; aged 71; died, March 25, 1953, of arteriosclerotic heart disease.

**Butler, John Dennison,** Newmarket, N. H.; B.M.C., class of 1907; aged 72; died, March 5, 1953.

**Comerer, Jacob Alvin,** Allentown, Pa.; B.M.C., class of 1897; aged 85; died, September 5, 1953, of arteriosclerosis.

**Davies, George William,** Verona, N. J.; B.M.C., class of 1905; served during World Wars I and II; aged 74; died, May 24, 1953, of carcinoma of the esophagus and cardiac decompensation.

**Feman, Jacob George,** Keansburg, N. J.; class of 1930; aged 48; died, June 9, 1953, of carcinoma of the colon.

**Fortney, Frank Dennis,** Beckley, W. Va.; B.M.C., class of 1904; aged 80; died, July 30, 1953, of cerebral hemorrhage.

**Friedman, Paul Norman,** Baltimore, Md.; class of 1943; aged 33; died, July 5, 1953, of ulcerative colitis.

**Gano, Charles Harkness,** Pittsburgh, Pa.; P & S, class of 1911; aged 68; died, August 13, 1953, of cerebrovascular accident.

**Gorman, John Joseph,** Fall River, Mass.; P & S, class of 1908; aged 67; died, May 28, 1953, of cerebral hemorrhage and bronchopneumonia.

**Griffith, William Allen,** Berwyn, Md.; P & S, class of 1909; aged 72; died, June 16, 1953, of cerebral hemorrhage.

**Haberer, Herman Joseph,** Utica, N. Y.; P & S, class of 1906; aged 72, died, May 30, 1953, of congestive heart failure.

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*Bulletin* OF
THE SCHOOL OF MEDICINE
*UNIVERSITY OF MARYLAND*

VOLUME 39 *April, 1954* NUMBER 2

# EFFECTS OF ACUTE DECOMPRESSION STRESS UPON PLASMA ELECTROLYTES AND RENAL FUNCTION IN ANESTHETIZED DOGS*

DIETRICH C. SMITH AND FREDERICK P. FERGUSON

With the technical assistance of

CAROLYN F. HENDRICKSON AND JEANNE Q. BARRY

DEPARTMENT OF PHYSIOLOGY, UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE, BALTIMORE, MARYLAND

In a previous communication Ferguson and Smith (1) reported the effects of acute decompression stress upon the distribution of potassium and sodium in the unanesthetized dog. Further analysis of certain aspects of the problem required acute surgical procedures that could be done only under anesthesia. It therefore became desirable to know to what extent, if any, the responses seen in the unanesthetized animal were modified by anesthesia. It was also necessary to justify the use of data from the anesthetized animal in interpreting results obtained on an unanesthetized dog.

## METHODS

Sodium pentobarbital, Nembutal,® was administered parenterally, usually intraperitoneally, in doses of 35 mgm./kgm. of body weight. If anesthesia were not induced in 30 minutes or if signs of recovery were seen during the experiment, more anesthetic was administered, usually intravenously. All experiments were performed upon trained female dogs, thoroughly accustomed to the procedure and submitting willingly to it when conscious. Free access to 3 liters of a 9:1 mixture of water and evaporated milk on the afternoon preceding the experiment insured adequate hydration without diuresis the next morning.

The experimental procedure was essentially the same as that described in detail in a previous communication (1). Following anesthetization, blood and urine samples were collected during two successive 20-minute predecompression periods, with observation of precautions necessary for renal clearance studies. The dog was then placed in a decompression chamber and exposed to a simulated altitude of 30,000 feet for 30 minutes. At the end of this time the dog was returned to room atmospheric pressure for collection of blood and urine samples. Two more 30 minute decompression periods followed, with blood and urine collected at the end of each. The dog was then kept at room atmospheric pressure for 90 minutes (postdecompression period), at which time a blood sample was drawn and the experiment terminated. Chemical analyses and calculation of renal function data were performed as previously described (1).

---

* This investigation was supported by a grant from the Frank C. Bressler Reserve Fund of the University of Maryland School of Medicine.

Ten decompression and 9 control experiments were made upon 8 different dogs. In two decompression experiments the dogs died either during (experiment 26A) or immediately after (experiment 21A) exposure to altitude. Control experiments, sometimes referred to as the undecompressed series, were identical with the decompression experiments, except that the chamber was kept at room atmospheric pressure by proper setting of control valves while the evacuation pumps were operating. At least a week elapsed between experiments when a dog was used more than once.

## RESULTS

*Effect of Acute Decompression Stress upon Plasma Potassium:* Experiments dealing with the effects of decompression upon plasma potassium in anesthetized dogs are complicated by the fact that the anesthetic itself (Nembutal) has a depressing effect upon the potassium concentration. In the experimental series the mean preanesthesia level of potassium was 4.33 mEq./l. as compared to a level of 3.54 mEq./l. following Nembutal administration. In the control series, the mean value fell from 4.52 to 4.26 mEq./l. with anesthesia. (See table 1.) At present we have no explanation for the difference in degree of response shown by the two series.

Upon decompression to a simulated altitude of 30,000 feet, however, a further decrease in plasma potassium concentration occurred in the experimental series, the lowest mean value (3.09 mEq./l.) being reached during the third period of decompression (table 1). The effect was pronounced in every case, with the possible exception of experiment 68A, in which the decrease was not apparent until the third period. During the post-decompression phase, the mean concentration returned to 3.57 mEq./l., which corresponds closely to the predecompression level of 3.54 mEq./l. No downward trend was seen in the plasma potassium concentration in the undecompressed control series, during those periods in which the decompression occurred in the experimental series.

Ferguson and Smith (1) have reported a decrease in plasma potassium from a mean level of 4.09 to about 3.30 mEq./l. in unanesthetized dogs, decompressed to 30,000 feet. Qualitatively, the anesthetized animals respond in a similar manner although the mean decrease is somewhat less marked (11 vs. 19 per cent), presumably because the pre-decompression level has already been reduced by the anesthetic. It is of interest to note that the combined effect of anesthesia and decompression decreases the mean plasma potassium concentration by a total of 27 per cent.

*Effect of Acute Decompression Stress upon Sodium:* Inspection of table 1 reveals that the plasma sodium concentration remains unaffected by the anesthetic and also by exposure of the anesthetized animals to a simulated altitude of 30,000 feet. The stability of plasma sodium during decompression of unanesthetized dogs has been demonstrated in a previous communication (1).

TABLE 1

*Effects of Acute Decompression Stress (30,000 feet) upon Plasma Potassium and Plasma Sodium in Anesthetized Dogs**

| Dog No. | Exp. No. | Pre-decompression† mEq/1 Plasma | | Decompression Period I mEq/1 Plasma | | Decompression Period II mEq/1 Plasma | | Decompression Period III mEq/1 Plasma | | Post-decompression‡ mEq/1 Plasma | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | K | Na | K | Na | K | Na | K | Na | K | Na |
| 5 | 15A | 3.57 | 147 | 2.74 | 146 | 3.25 | 140 | — | — | 3.87 | 148 |
| 1 | 16A | 3.40 | 157 | 3.29 | 155 | 3.29 | 152 | 2.55 | — | 3.57 | 160 |
| 5 | 18A | 4.04 | 147 | 3.61 | 147 | 3.61 | 147 | 3.44 | 147 | 3.75 | — |
| 1 | 19A | 2.83 | 156 | 2.62 | 156 | 2.79 | 155 | 2.38 | 156 | 2.50 | 156 |
| 1 | 20A | 3.35 | 152 | 2.75 | 157 | 2.67 | 148 | 2.62 | 152 | 3.19 | 155 |
| 5 | 21A | 3.56 | 147 | 3.03 | 150 | 3.18 | 148 | 3.68 | 150 | — | — |
| 7 | 23A | 3.68 | 149 | 3.25 | 152 | 3.02 | 144 | 2.94 | 141 | 3.51 | 147 |
| 8 | 26A | 3.21 | 150 | 2.83 | 149 | 2.83 | 155 | — | — | — | — |
| 9 | 27A | 3.79 | 148 | 3.50 | 147 | 3.65 | 148 | 3.68 | 148 | 4.35 | 148 |
| 11 | 68A | 3.96 | 154 | 4.11 | 153 | 4.04 | 154 | 3.46 | 154 | 3.84 | 153 |
| Mean... | | 3.54 | 151 | 3.17 | 151 | 3.23 | 149 | 3.09 | 150 | 3.57 | 152 |
| S.E.§... | | ±0.11 | ±1.2 | ±0.16 | ±1.3 | ±0.15 | ±1.6 | ±0.20 | ±2.1 | ±0.20 | ±2.2 |
| Undecompressed Controls | | | | | | | | | | | |
| Mean... | | 4.26 | 151 | 4.22 | 151 | 4.28 | 151 | 4.50 | 152 | 4.67 | 152 |
| S.E..... | | ±0.13 | ±3.8 | ±0.09 | ±2.7 | ±0.12 | ±2.2 | ±0.11 | ±2.2 | ±0.18 | ±3.9 |

* Mean pre-anesthesia values for decompressed series: K = 4.33 ± 0.30, Na = 150 ± 1.4 mEq/1; for undecompressed control series: K = 4.52 ± 0.11, Na = 152 ± 4.9 mEq/1 plasma.

† Pre-decompression values represent averages calculated from two blood samples taken 20 min. apart.

‡ Post-decompression values obtained from sample taken 90 min. after end of third decompression period.

§ Standard error of mean.

*Effect of Acute Decompression Stress upon the Hematocrit:* Preanesthesia hematocrit values were determined in each of the 9 control experiments and in 4 of the 10 decompression experiments. With one exception (experiment 26A, in which the dog died during decompression), the anesthetic produced a marked decrease in the hematocrit. The mean value declined from 50 to 41.1 in the experimental series and from 45.6 to 39.4 in the undecompressed controls (figure 1).

Upon decompression, a rise in the hematocrit occurred in 9 of the 10 experiments, the exception again being experiment 26A. A maximum mean value of 45.8 was observed (figure 1). During the post-decompression phase, the mean value rose to 49.5, an increase which is attributed to the partial recovery of the animals from the anesthetic. In the undecompressed control series, the hematocrit values remained relatively constant, except for the final sample. In this sample a slight rise was noted, again as a probable result of partial recovery from the anesthesia.

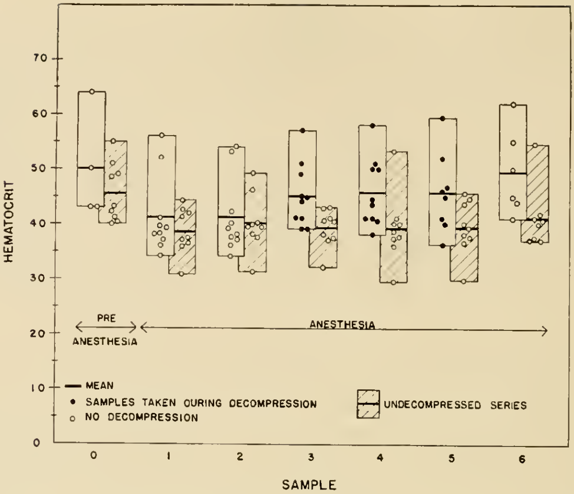

FIG. 1. Effect of acute decompression stress (30,000 feet) upon the hematocrit in anesthetized dogs. Each circle represents a single blood sample.

Qualitatively the hematocrit response of the anesthetized dog to decompression agrees with that of the unanesthetized animal (1). In the latter, the increase was of the order of 4.3 per cent (from a pre-decompression level of 46.4 to a level of 48.4 during decompression), whereas in the anesthetized series it was about 11 per cent (from 41.1 to 45.6). Presumably the augmented response in the anesthetized dogs is related to the initial depression of the hematocrit produced by the anesthetic.

*Effect of Acute Decompression Stress upon Urine Flow:* Table 2 shows that decompression to 30,000 feet failed to produce any consistent effect upon the rate of urine flow in anesthetized dogs. With the exception of experiment 23A, in which a marked increase was noted during decompression, changes which occurred were slight and random. The mean rate of flow prior to decompression was 0.19 cc. per minute and that during decompression was 0.20 cc. per minute. In the undecompressed controls, the mean urine flow appears to decrease considerably during the last period. This does not represent a general response. It is mainly the result of a marked oliguria in two animals which had shown relatively high rates of flow during the early periods.

These data illustrate two interesting features when compared with those reported upon unanesthetized dogs which were similarly hydrated (1). First, the anesthetic apparently depresses the rate of urine formation. Since the average rate of flow for unanesthetized animals (experimental and control series) during the pre-decompression periods was 0.46 cc. per minute, that for the anesthetized animals (experimental

TABLE 2

*Effects of Acute Decompression Stress (30,000 feet) upon Glomerular Filtration Rate and Urine Flow in Anesthetized Dogs (cc/min)*

| Dog No. | Exp. No. | Pre-decompression* | | Decompression | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | Period I† | Period II | | Period III | |
| | | G.F.R. | Urine flow | Urine flow | G.F.R. | Urine flow | G.F.R. | Urine flow |
| 5 | 15A | 40.5 | 0.19 | 0.11 | 34.4 | 0.19 | — | — |
| 1 | 16A | 25.0 | 0.12 | 0.03 | 29.2 | 0.30 | 19.4 | 0.14 |
| 5 | 18A | 36.9 | 0.22 | 0.17 | 35.2 | 0.22 | 36.9 | 0.20 |
| 1 | 19A | 26.9 | 0.23 | 0.20 | 20.3 | 0.19 | 27.4 | 0.19 |
| 1 | 20A | 34.1 | 0.32 | 0.30 | 37.0 | 0.64 | 32.1 | 0.30 |
| 5 | 21A | 42.1 | 0.44 | 0.52 | 24.9 | 0.19 | 17.1 | 0.22 |
| 7 | 23A | 27.5 | 0.06 | 0.30 | — | — | 36.8 | 0.16 |
| 8 | 26A | 33.4 | 0.11 | 0.13 | 15.5 | 0.09 | — | — |
| 9 | 27A | 23.6 | 0.12 | 0.11 | 22.4 | 0.14 | 23.0 | 0.16 |
| 11 | 68A | 21.1 | 0.11 | 0.12 | 25.4 | 0.12 | 21.3 | 0.08 |
| Mean | | 31.1 | 0.19 | 0.20 | 27.1 | 0.23 | 26.8 | 0.18 |
| S.E. | | ±2.33 | ±0.04 | ±0.04 | ±2.51 | ±0.06 | ±2.68 | ±0.02 |
| Undecompressed Controls | | | | | | | | |
| Mean | | 23.2 | 0.26 | 0.24 | 24.7 | 0.21 | 22.7 | 0.12 |
| S.E. | | ±1.73 | ±0.12 | ±0.11 | ±1.80 | ±0.07 | ±2.22 | ±0.01 |

* Pre-decompression values represent averages calculated from two consecutive 20 min. urine samples.

† Glomerular filtration rate was not determined for this period.

and control) is only 0.23 cc. per minute. Secondly, whereas the urine flow of anesthetized dogs is not appreciably altered by decompression to 30,000 feet, that of the unanesthetized animals was reduced by an average of about 43 per cent.

*Effect of Acute Decompression Stress upon Glomerular Filtration Rate:* With the possible exception of two experiments (21A and 26A), there is no evidence that glomerular filtration rate was significantly altered by exposure to 30,000 feet (table 2). In these two cases there was a decrease. In both instances, however, the dogs died while the experiment was in progress. It therefore seems probable that the depressed filtration rate in these two dogs reflects a progressive deterioration in the general condition of the animals rather than a specific effect of decompression. Inspection of the undecompressed control data shows no significant trend in glomerular filtration rate, the range throughout being from 23.2 to 24.7 cc. per minute.

In the unanesthetized dogs (1) there was no suggestion of any change in glomerular filtration rate during decompression. Nor is there a significant difference between the pre-decompression filtration rate in this group and that seen in the anesthetized series. Thus the reduced urine flow seen before decompression in the anesthetized animal is probably the result of an increased tubular reabsorption of water.

*Effect of Acute Decompression Stress upon Sodium Excretion:* Table 3 shows that decompression of anesthetized dogs resulted in an increased excretion of sodium in

TABLE 3

*Effect of Acute Decompression Stress (30,000 feet) upon Sodium Excretion in Anesthetized Dogs*

| Dog No. | Exp. No. | Pre-decompression* | | | | | Decompression† | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Period II | | | | | Period III | | | | |
| | | Clearance | Filtered | Excreted | Reab-sorbed | Per cent of filtered reab-sorbed | Clearance | Filtered | Excreted | Reab-sorbed | Per cent of filtered reab-sorbed | Clearance | Filtered | Excreted | Reab-sorbed | Per cent of filtered reab-sorbed |
| | | cc/min | μ Eq/min | | | | cc/min | μ Eq/min | | | | cc/min | μ Eq/min | | | |
| 5 | 15A | 0.52 | 5926 | 19.4 | 5907 | 99.68 | 0.25 | 4925 | 35.2 | 4890 | 99.29 | — | — | — | — | — |
| 1 | 16A | 0.17 | 3916 | 26.1 | 3890 | 99.34 | 0.38 | 4474 | 58.7 | 4415 | 98.68 | — | — | — | — | — |
| 5 | 18A | 0.10 | 5406 | 14.0 | 5392 | 99.74 | 0.25 | 5160 | 36.2 | 5124 | 99.30 | 0.27 | 5411 | 39.3 | 5372 | 99.28 |
| 1 | 19A | 0.23 | 4194 | 35.6 | 4158 | 99.14 | 0.33 | 3149 | 51.0 | 3098 | 98.38 | 0.27 | 4264 | 42.3 | 4222 | 99.02 |
| 1 | 20A | 0.14 | 5193 | 20.8 | 5173 | 99.61 | 0.09 | 5652 | 14.0 | 5638 | 99.75 | 0.49 | 4822 | 73.5 | 4749 | 98.49 |
| 5 | 21A | 0.38 | 6169 | 56.3 | 6112 | 99.08 | 0.23 | 3704 | 34.9 | 3669 | 99.06 | 0.14 | 2545 | 21.4 | 2524 | 99.17 |
| 7 | 23A | 0.01 | 4067 | 2.1 | 4065 | 99.95 | — | — | — | — | — | 0.20 | 5241 | 27.8 | 5213 | 99.47 |
| 8 | 26A | 0.04 | 4993 | 5.3 | 4988 | 99.90 | 0.09 | 2351 | 12.9 | 2338 | 99.45 | — | — | — | — | — |
| 9 | 27A | 0.01 | 3484 | 1.9 | 3482 | 99.94 | 0.04 | 3313 | 5.9 | 3307 | 99.82 | 0.07 | 3407 | 10.7 | 3396 | 99.68 |
| 11 | 68A | 0.04 | 3232 | 6.6 | 3225 | 99.78 | 0.03 | 3896 | 5.1 | 3891 | 99.87 | 0.03 | 3278 | 4.1 | 3274 | 99.88 |
| Mean | | 0.16 | 4658 | 18.8 | 4639 | 99.62 | 0.19 | 4069 | 28.2 | 4041 | 99.29 | 0.21 | 4138 | 31.3 | 4107 | 99.28 |
| S.E. | | ±0.05 | ±323 | ±5.4 | ±320 | | ±0.04 | ±356 | ±6.5 | ±355 | | ±0.06 | ±412 | ±8.8 | ±407 | |
| Undecompressed Controls | | | | | | | | | | | | | | | | |
| Mean | | 0.07 | 3496 | 11.2 | 3485 | 99.72 | 0.08 | 3711 | 12.1 | 3699 | 99.71 | 0.06 | 3425 | 9.3 | 3416 | 99.75 |
| S.E. | | ±0.03 | ±255 | ±4.2 | ±252 | | ±0.03 | ±283 | ±4.7 | ±279 | | ±0.03 | ±321 | ±3.7 | ±318 | |

* Pre-decompression values represent averages calculated from two consecutive 20 min. clearance periods.

† Clearances were not run during first decompression (Adaptation) period.

8 of the 10 experiments. The exceptions were experiment 21A, in which the animal died immediately after the third decompression period, and experiment 68A. (In experiment 20A increased sodium excretion failed to occur in Period II.) It is interesting to note further that in experiment 21A the pre-decompression level of sodium excretion was unusually high. The mean rate of sodium excretion rose from a pre-decompression level of 18.8 μEq./minute to 28.2 and 31.3 μEq./minute, during the second and third periods of decompression, respectively. During the first period of decompression (not included in the table) the rate averaged 22.5 μEq./minute. In the undecompressed controls, sodium excretion remained relatively constant as indicated by the mean values at the bottom of the table.

The difficulties involved in separating the influence of changes in filtration from the influence of changes in tubular reabsorption in explaining alterations of sodium excretion have been pointed out by Berliner (2) and others. Careful examination of table 3 reveals that in a few periods in which augmented sodium excretion occurred the effect was the result of the fact that the amount filtered increased more than the amount reabsorbed, rather than to an actual depression of the reabsorption mechanism (Period II of experiment 16A, Period III of experiments 19A and 23A). In most instances, however, the increased sodium excretion cannot be explained upon the basis of increased filtration. In three periods (Period II of experiments 15A, 19A and 26A), it occurred in spite of a marked decrease in sodium filtration. In four (Period II of experiments 18A and 27A and Period III of experiments 20A and 27A), it occurred in the presence of a probable decrease*, and in one (Period III of experiment 18A) in the presence of no detectable change in sodium filtration. On the basis of these data, it appears that the increased sodium excretion in anesthetized dogs during decompression is usually the result of decreased tubular reabsorption.

In unanesthetized dogs, the effects of decompression upon sodium excretion were not consistent (1). Furthermore, when an increase did occur in these animals, it was not possible to eliminate augmented sodium filtration as a possible contributing factor.

*Effect of Acute Decompression Stress upon Potassium Excretion:* As seen in table 4, potassium excretion increased in the anesthetized dog during decompression. This effect was produced in 7 of the 10 experiments, the 3 exceptions being 21A and 26A (which involved the two animals that died) and experiment 23A. The mean rate of excretion rose from a pre-decompression level of 10.59 μEq./minute to 15.94 and 13.47 μEq./minute. During the first decompression period, which is not included in the table, the rate was 17.12 μEq./minute.

Examination of table 4 also indicates that, with few exceptions (Period II of experiments 16A and 68A), the increased potassium excretion occurred in spite of a decreased potassium filtration. This indicated that it was the result of altered tubular function. Whether the mechanism involved is a decreased reabsorption or an increased secretion of potassium, or both, cannot be ascertained at present. The decreased potassium filtration is caused primarily by the depression of plasma potassium concentration in the presence of a relatively constant glomerular filtration rate (tables 1 and 2).

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* It must be kept in mind that an error of 1 cc./min. in the calculation of glomerular filtration rate will result in an error of the order of 150μ Eq./min. in sodium filtration.

## TABLE 4

*Effect of Acute Decompression Stress (30,000 feet) upon Potassium Excretion in Anesthetized Dogs*

| Dog No. | Exp. No. | Pre-decompression* | | | | Decompression† | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Period II | | | | Period III† | | | |
| | | Clearance | Filtered | Excreted | Per cent of filtered excreted | Clearance | Filtered | Excreted | Per cent of filtered excreted | Clearance | Filtered | Excreted | Per cent of filtered excreted |
| | | cc/min | μEq/min | | | cc/min | μ Eq/min | | | cc/min | μ Eq/min | | |
| 5 | 15A | 2.32 | 144.36 | 8.28 | 5.74 | 3.96 | 103.29 | 11.88 | 11.50 | — | — | — | — |
| 1 | 16A | 2.26 | 84.80 | 7.66 | 9.03 | 4.84 | 95.97 | 15.93 | 16.60 | 4.03 | 56.53 | 11.76 | 20.80 |
| 5 | 18A | 1.84 | 148.98 | 7.42 | 4.98 | 6.46 | 127.07 | 23.32 | 18.35 | 5.23 | 130.29 | 18.46 | 14.17 |
| 1 | 19A | 3.46 | 76.11 | 9.80 | 12.88 | 3.45 | 54.72 | 9.32 | 17.03 | 6.13 | 71.02 | 15.88 | 22.36 |
| 1 | 20A | 7.18 | 114.16 | 24.05 | 21.07 | 12.52 | 100.38 | 33.92 | 33.79 | 7.92 | 84.96 | 21.00 | 24.72 |
| 5 | 21A | 7.85 | 149.78 | 27.93 | 18.65 | 4.53 | 77.31 | 14.10 | 18.24 | 3.56 | 58.52 | 12.21 | 20.86 |
| 7 | 23A | 0.99 | 100.99 | 3.65 | 3.61 | — | — | — | — | 0.93 | 109.60 | 2.77 | 2.53 |
| 8 | 26A | 2.09 | 107.34 | 7.01 | 6.53 | 2.39 | 43.84 | 6.75 | 15.40 | — | — | — | — |
| 9 | 27A | 1.48 | 89.45 | 5.64 | 6.31 | 3.72 | 80.08 | 13.30 | 16.61 | 4.70 | 84.25 | 17.22 | 20.44 |
| 11 | 68A | 1.12 | 83.44 | 4.41 | 5.29 | 3.66 | 103.55 | 14.93 | 14.42 | 2.26 | 79.99 | 8.48 | 10.60 |
| Mean | | 3.06 | 109.94 | 10.59 | 9.41 | 5.06 | 87.36 | 15.94 | 17.99 | 4.35 | 84.40 | 13.47 | 17.06 |
| S.E. | | ±0.78 | ±9.01 | ±2.64 | ±1.93 | ±1.00 | ±8.69 | ±2.72 | ±2.10 | ±0.77 | ±8.83 | ±2.09 | ±2.63 |
| Undecompressed Controls | | | | | | | | | | | | | |
| Mean | | 1.43 | 99.89 | 6.34 | 5.57 | 1.60 | 105.62 | 6.92 | 6.12 | 2.01 | 101.35 | 9.16 | 8.49 |
| S.E. | | ±0.52 | ±9.02 | ±2.30 | ±1.88 | ±0.51 | ±9.64 | ±2.32 | ±1.50 | ±0.49 | ±11.68 | ±2.43 | ±1.63 |

* Pre-decompression values represent averages calculated from two consecutive 20 min. clearance periods.

† Clearances were not run during first decompression (Adaptation) period.

In the unanesthetized dog a similar increase in potassium excretion was seen during decompression to 30,000 feet (1). Since the effect occurred simultaneously with a decreased potassium filtration, it was attributed, as in the anesthetized dog, to an alteration of tubular function.

## DISCUSSION

A discussion of the results is complicated by the fact that sodium pentobarbital itself produces changes in the plasma potassium concentration and the hematocrit. While Stewart (3) reported the effects of sodium pentobarbital anesthesia on plasma potassium to be minimal in the dog, others observed a fall (4) (5) and still others a rise (6). Dial is said to produce a marked fall in the plasma potassium in the rabbit (3) (7) and in the dog (3). Larson and Brewer (5) observed that the decrease in serum potassium seen after sodium pentobarbital still occurred in the adrenal-demedullated dog.

A fall in hematocrit after sodium pentobarbital has been reported in the dog (8) (9) (10), in the cat (11) and the rat (12). This is probably caused by hemodilution resulting from relaxation of the spleen under the influence of the anesthetic and its consequent engorgement with red cells. Carr and Essex (9) observed no fall in hematocrit after sodium pentobarbital in the splenectomized dog. Plasma volume in the dog is said to increase after sodium pentobarbital (10) (13), although total blood volume remains constant. Increase in plasma volume in the cat has also been noted after sodium pentobarbital (14).

The depression of urine flow by barbiturates has been previously noted in the dog and rabbit (3) (15) (16). Since anesthesia does not appear to depress glomerular filtration rate, the decrease in urine flow in the dogs in this series would seem to be the result of increased tubular reabsorption of water.

There are no reports in the literature concerning the behavior of either the plasma potassium or the hematocrit in animals subjected to decompression under barbiturate anesthesia. This is not the case concerning urine flow. Most investigators (17) (18) (19) (20) report a decrease in urine volume on exposure to low oxygen tension in dogs and rabbits under a wide variety of barbiturates. This is contrary to the observations reported here, where decompression is found to produce no change in the already depressed urine flow of the anesthetized dog. There seems to be no obvious explanation for the difference in results, since all experiments were executed under comparable degrees of hydration and anoxia and with similar anesthetic agents.

Selkurt (21), however, has recently reported an increase in urine flow from the kidney of a barbitalized dog perfused with venous blood. He ascribed this increase to a direct impairment of tubular function by hypoxia, since humoral factors were virtually excluded from the experiment. In addition the increase in urine flow exceeded that required as a result of the increase in sodium excretion. Kline (22) also reported a polyuria in cats anesthetized with dial, after 90 minute exposure to 28,000 feet.

The decrease in plasma potassium during hypoxia in the unanesthetized dog cannot be accounted for by the increased potassium excretion (1). Such would also appear to be the case in the anesthetized dog, for in three of the preceding experiments plasma potassium fell when urinary potassium excretion decreased (experiments 21A, 23A and 26A). In one instance (experiment 20A) the increase in potassium excretion during decompression was so slight as to be of no significance in explaining the substantial fall in plasma concentration. Kline (22) noted a fall in plasma potassium after decompression in the nephrectomized cat, anesthetized with dial.

Although decreases in the excretion of potassium during decompression have been noted occasionally, the usual response is an increase—a response which is true for sodium as well. Selkurt (21) also reported increases in the excretion of potassium and sodium from the kidney of a dog anesthetized with sodium pentobarbital and perfused with venous blood. Kline (22) reported a 60 per cent increase in the excretion of potassium and a 100 per cent increase in the excretion of sodium in cats anesthetized with dial and decompressed to 28,000 feet for 90 minutes.

While relatively severe hypoxia will decrease plasma potassium in the cat, still more severe hypoxia, leading to respiratory collapse, will increase the potassium (22) (23). In the unanesthetized dog a similar relationship apparently exists. Most workers report a decrease in plasma potassium during hypoxia (1) (24) (25), although one group of investigators noted a rise (4). If, however, exposure is increased to the point of respiratory collapse, the plasma potassium in the dog rises (Ferguson and Smith, unpublished observations). Since then the rise in plasma potassium noted in the cat and dog at respiratory collapse is a terminal sign, the fall in plasma potassium noted in the anesthetized dog indicates a degree of stress which is well tolerated. This is true in spite of the fact that anesthetization with sodium pentobarbital in the cat sharply decreases the survival time of cats in 5 per cent oxygen (26).

Thus the fall in plasma potassium, in the dog anesthetized with sodium pentobarbital, together with other effects of acute decompression stress to 30,000 feet indicates that the anesthesia has not so altered the physiologic state of the animal. Therefore comparison with the unanesthetized animal is not difficult or futile.

## SUMMARY

The effects of hypoxia, produced by decompression to a simulated altitude of 30,000 feet for 90 minutes on the plasma concentration and excretion of sodium and potassium have been studied in dogs anesthetized with sodium pentobarbital. Anesthetized controls were subjected to the same experimental procedure except for decompression.

Plasma potassium concentration was initially decreased by the anesthesia. A further depression (averaging about 11 per cent) occurred during the first 30 minutes of decompression and was maintained throughout the remainder of the exposure. Plasma sodium concentration remained unchanged by the anesthesia and the subsequent decompression.

The hematocrit value was depressed initially by the anesthesia, but increased during the first 30 minutes of decompression. It remained elevated throughout the duration of the exposure.

In the kidney neither the glomerular filtration rate nor urine flow showed any significant change during decompression. The excretion of sodium and potassium was somewhat elevated. In the case of sodium the increased excretion was apparently the result of a decreased tubular reabsorption, since it often occurred in the presence of reduced sodium filtration. The increase in potassium excretion also occurred simultaneously with decreased filtration and is, therefore, caused by decreased tubular reabsorption, increased tubular secretion, or both.

It is concluded that, under the conditions of these experiments, the response of the anesthetized dog to acute decompression stress is similar to that of the conscious animal with respect to the hematocrit, plasma sodium and potassium, glomerular filtration rate and potassium excretion. Unlike the conscious animal, the anesthetized dog shows no consistent depression of urine flow and an increased sodium excretion upon decompression.

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# REGENERATION OF SYMPATHETIC NERVES—AN EXPERIMENTAL STUDY*

CALVIN Y. HADIDIAN, M.D. AND OTTO C. BRANTIGAN, M.D.

Since the inauguration of surgical treatment as applied to the sympathetic nervous system, attention has been centered on the problems raised by regeneration of the nerves. The frequent incidence of this undesirable end result and the relative speed with which it occurs have created a challenge to surgeons.

Although regeneration of the peripheral nerves, both somatic and autonomic, is an accepted phenomenon, its real nature is still debatable. It is accomplished by the growth of neurofibrils from the proximal segment. The neurileminal sheaths on both ends grow towards each other, thus providing a matrix for the newly growing neurofibrils. The rate of growth of somatic nerves has been variously estimated at 1.0 to 4.5 mm. per day[6]. There seems to be considerable confusion and uncertainty with regard to the true nature of this so-called "neurotropism."

The extent and manner of regeneration have been studied in the autonomic nervous system, especially the sympathetic nerves (dorsolumbar outflow), with essentially similar results. Haimovici and Hodes[5] in a series of experiments on extensively sympathectomized cats made note of this factor. They sympathectomized cats from the stellate to the pelvic ganglia and sacrificed them 54 to 236 days after the final operation. The cephalic ends of the intact sympathetic trunks were stimulated electrically and produced signs of sympathetic regeneration in 6 out of 7 animals, namely, retraction of the nictitating membrane and dilatation of the pupil.

It is evident that the problem of regeneration of autonomic nerves becomes specially vital to the surgery of that system—dorsal, dorsolumbar or lumbar. Permanent results usually are desired and for this many methods for the prevention of functional resumption by regeneration have been used.

Smithwick[2] in his outstanding contribution to this field of surgery has advocated the technic of preganglionic resection or extraspinal root avulsion to insure as near a complete lack of regeneration as possible. He has also recommended ligation of the distal cut end of the trunk and wrapping of a silk cylinder over the proximal end. White and Hamlin[1] have worked with tantalum sleeves and find their use more satisfactory in avoiding regeneration.

In an effort to study the extent, nature and methods of prevention of regeneration of the sympathetic nerves in animals, a limited experiment was carried out in the laboratories of the Department of Experimental Surgery of the University of Maryland School of Medicine.

## MATERIAL AND METHODS

Ten dogs of varying sizes were used in this experiment. They were anesthetized with intravenous injections of nembutal and intubated. Air by intermittent positive

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pressure was administered by means of a special machine. The thorax was entered through the left side in the usual manner. The sympathetic trunk was identified and a section measuring approximately one inch was excised to include one segmental ganglion. The cut ends were treated as follows: in three the ends were left free; two were ligated with silk and one had phenol injection. In two instances the free ends were interposed between the neighboring intercostal group of muscles, thus changing the direction of their course. One received an injection of 95 per cent alcohol. The last animal had a small piece of sea sponge inserted between the cut ends.

The animals were biopsied at the end of four months, except for two dogs that died early in their postoperative course. The gross impression of regeneration between the cuts ends was considered rather crude because of the presence of fibrous tissue reaction. Therefore the terminal ends of the trunk as well as the intervening tissues were removed for microscopic studies. The presence of neurofibrils in this intervening tissue was regarded as evidence of regeneration.

## RESULTS

One dog with muscle interposition died of pneumonia on the nineteenth postoperative day. Another dog, with the ends left free, died on the twelfth day and was not autopsied. Of the remaining eight dogs, the following results were obtained:

| | Number of Dogs | Evidence of Regeneration |
|---|---|---|
| Ends left free | 2 | Present |
| Muscle interposition | 1 | Absent |
| Phenol injection | 1 | Present |
| Alcohol injection | 1 | Absent |
| Sponge interposition | 1 | Present |
| Silk ligation | 2 | Absent |

## DISCUSSION

Although this series is limited in number, several factors are worthy of comment. Three of the four dogs in whom the trunk ends were left free and facing each other, with or without injection, showed evidence of regeneration. Also, it appears from this and other studies that changing the direction of the cut ends of the trunk is deleterious to proper regeneration. This procedure was followed in the animal with intercostal muscle interposition. Furthermore, it is obvious that the mere presence of a barrier between the cut ends without changing their direction does not hamper regrowth. In fact, it is probable that such a foreign body of the porous type may help in the growth of the neurofibrils.

Finally and significantly, neither of the two animals in whom the trunk ends were ligated with silk showed any attempt at bridging the gap.

## CONCLUSION

It is fair to assume that the end results of surgery of the peripheral sympathetic nervous system will depend upon the permanency of the denervation of the organs or extremities involved. To achieve this end various methods have been tried and advocated. It appears from the present study, that ligation with silk of all cut nerve ends and the deflection of the cut ends of the main trunks form a safeguard that can be adopted without undue effort. A larger series of similar studies with a long term follow-up is necessary to reach a more definite conclusion. Such a study is under way at the present time in the same laboratories.

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## ERROR

In the October, 1953 number of the Bulletin (Vol. 38, No. 4), Figures 1 and 2 on page 165 are reversed in position. The figure describing plasma potassium changes should be in the upper part of the page, labeled as Fig. 1; that describing hematocrit changes should be in the lower part of the page, labeled as Fig. 2.

# MAGNAMYCIN OINTMENT IN THE TREATMENT OF THE PYODERMAS*

*(Preliminary Report)*

HARRY M. ROBINSON, JR., M.D., DAVID BACHARACH, M.D. AND JOHN STRAHAN, M.D.

A clinical evaluation of each new antibiotic is an absolute necessity before the preparation can be produced for general usage. Magnamycin®, which is isolated from the elaborated products of the mold streptomyces halstedii, is a crystalline monobasic substance with antibiotic properties.

This report is a statistical evaluation of the effect of 3 per cent Magnamycin ointment in the treatment of pyodermas and other dermatoses complicated by secondary pyogenic infection.

### *Patients Studied*

The patients included in this study were obtained from our private practices and the Dermatology Clinic of the University Hospital. In view of the fact that the results were the same, no attempt has been made to separate these individuals into race, sex, or age groups. Fifty nine of these patients had primary pyodermas and 22 had other dermatoses complicated by secondary pyogenic infection.

### *Preparation Used*

A 3 per cent ointment of Magnamycin was prepared in a base consisting of petrolatum and mineral oil.

### *Method of Treatment*

The ointment was applied three times a day after crusts had been removed with warm water.

### *Result of Treatment*

The cases of impetigo contagiosa and ecthyma responded slowly to Magnamycin ointment. There was complete resolution of the impetigo contagiosa lesions in 5 to 38 days. The time required for involution varied with the extent of the lesions. Thirteen cases of ecthyma healed in from 12 to 37 days, depending on the severity of the condition. Two cases of sycosis vulgaris showed complete healing in 12 and 28 days respectively. The third case of sycosis vulgaris did not respond. Four cases of folliculitis were healed in from 7 to 14 days. Three cases of ecthyma developed local irritation from the application of the Magnamycin ointment. These patients were not sensitive to Magnamycin.

### *Conditions Other than Pyodermas Complicated by Secondary Infection*

Magnamycin ointment was applied locally to 22 patients with primary dermatoses complicated by secondary pyogenic infection. The secondary infection resolved in

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CHART I

*Treatment of Pyodermas*

| Diagnosis | No. of Cases | Shortest Time to Heal Lesions | Longest Time to Heal Lesions | Adverse Reactions | Failures |
|---|---|---|---|---|---|
| | | *days* | *days* | | |
| Impetigo contagiosa | 38 | 5 | 38 | 0 | 0 |
| Ecthyma | 13 | 12 | 37 | 3 (local irritation) | 0 |
| Furunculosis | 1 | — | — | — | 1 |
| Sycosis vulgaris | 3 | 12 | 28 | 0 | 1 |
| Folliculitis | 4 | 7 | 14 | 0 | |

CHART II

*Dermatoses Complicated by Secondary Infection*

| Diagnosis | No. of Cases | Days Necessary to Eradicate Secondary Infection | | Adverse Reaction | Failure |
|---|---|---|---|---|---|
| | | Shortest | Longest | | |
| Infectious eczematoid dermatitis | 1 | 8 | | 0 | 0 |
| Infected dermatophytosis | 4 | 5 | 6 | 0 | 0 |
| Infected seborrheic dermatitis | 6 | 7 | 18 | 0 | 0 |
| Tinea cruris infected | 1 | 7 | | | |
| Eczema, secondarily infected | 4 | 8 | 17 | 0 | 0 |
| Contact dermatitis secondarily infected | 5 | 9 | 19 | 0 | 0 |
| Tinea capitis, secondarily infected | 1 | | 25 days failure | | |

from 5 to 25 days. The case of tinea capitis with secondary infection was a treatment failure after 25 days of ointment application. Magnamycin ointment produced no improvement in any of the underlying dermatoses.

## *Conclusions*

1. Eighty one patients with dermatoses were treated with Magnamycin ointment. Fifty nine had primary pyodermas and 22 had dermatoses with secondary pyogenic infection.

2. Three per cent Magnamycin ointment produced involution of the pyodermas in 5 to 38 days.

3. Three per cent Magnamycin ointment caused rapid involution of the secondary pyogenic infection complicating dermatoses but did not improve the primary condition.

4. Three cases of local irritation were noted in the series.

5. The results of therapy with this antibiotic ointment are inferior to those obtained with Terramycin®, Aureomycin® and Neomycin® ointments.

# ADRENAL STEROIDS AND CEREBROVASCULAR PERMEABILITY—PRELIMINARY NOTE

R. G. GRENELL, PH.D. AND J. MENDELSON†

In 1947 the studies of Prados, Strowger and Feindel (1) were repeated and extended by Grenell and McCawley (2). It was shown at that time that adrenal cortical extract can pass across the blood-brain barrier cnd produce characteristic electroencephalographic changes. Similar observations relative to alterations in the electroencephalographic pattern are made for desoxycorticosterone acetate (DCA), although such changes were qualitatively different from those observed following administration of adrenal cortical extract. In addition, it was demonstrated that adequate doses of adrenal cortical extract (ACE) protected the cerebral cortex from experimentally induced exposure edema. Some indications were also obtained that were suggestive of marked therapeutic efficacy of ACE in treatment of post-traumatic concussion.

The increase in availability of the adrenal steroids over the last few years prompted the present study of the relationship of these compounds to experimental cerebral exposure edema.

Twelve adult white rabbits have been employed so far in this study. Under local (1 per cent procaine) anesthesia the cerebral cortex was exposed to the air by making a trephine hole ½ inch in diameter and lifting the underlying dura. The operation was preceded and followed by injections as noted in Table 1. Observations were then made of the degree of cortical bulging through the trephine hole and the character of the cerebral pulsations. Before sacrificing each animal injections of 1 per cent trypan blue (10 cc/kg) were made into the ear veins. Animals were sacrificed by air embolism and the brains were removed and examined for trypan blue diffusion.

As noted in Table 1 the control animals exhibited gross bulging of cortical tissue and loss of cortical pulsations in 3–6 hours. In addition, the trypan blue administered prior to sacrificing the animals had diffused into the cortex. Under normal physiologic conditions trypan blue does not pass the blood-brain barrier. Adrenal cortical extract (ACE) and 17-Hydroxycorticosterone gave excellent protection up to 10–12 hours and no diffusion of trypan blue was observed. Cortisone Acetate and ACTH gave only fair and poor protection respectively while Desoxycorticosterone Acetate **increased** the degree of vascular embarrassment.

The protection afforded cerebral vascular permeability by injections of adrenal cortical extract prior to exposure of the cerebral cortex has been reconfirmed. It is most interesting that 17-Hydroxycorticosterone is as effective as the total extract. There appears to be a definite difference in the action of the 11-oxysteroids and DCA on cerebro-vascular permeability. Further experiments are to be carried out. These results suggest anew the therapeutic use of adrenal cortical extract and 17-Hydroxycorticosterone in clinical entities manifesting symptomatology of cerebral edema. The clinical use of ACE is definitely suggested in neurosurgical procedures and post-traumatic situaticns where cerebral edema is a potential complication.

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TABLE 1

| GROUP | STEROID INJECTED | DOSAGE | INJECTION TIME | OBSERVATION |
|---|---|---|---|---|
| Control | Saline | 2 cc/kg I.M. | ½ hour prior to performing operation | Gross cortical bulging in 3-6 hours. Loss of cortical pulsations in 3 hours. Trypan blue diffusions through cortex |
| Experimental 1 | Desoxycorticosterone acetate (DCA) (Percortin—Ciba) | 2 mg/kg I.M. | 1 hour prior to performing operation. Booster dose immediately after operation | Gross cortical bulging in 3 hours. Loss of pulsations after 1 hour. Gross trypan blue diffusion through cortex |
| Experimental 2 | 17-Hydroxycorticosterone acetate (Hydrocortone-Merck) | 25 mg/kg I.M. | 1 hour prior to performing operation. Booster dose 3½ hours after operation | No loss of cortical pulsations at 12 hours. No cortical bulging at 10-12 hours. No trypan blue diffusion into cortex |
| Experimental 3 | Adrenal cortical extract (Upjohn) | 2 cc/kg I.M. | ½ hour prior to performing operation. Booster dose 3½ hours after operation | No loss of cortical pulsations at 10 hours. No cortical bulging at 10 hours. No trypan blue diffusion into cortex |
| Experimental 4 | Cortisone acetate (Cortone-Merck) | 25 mg/kg I.M. | ½ hour before performing operation. Booster dose 3 hours after operation | No cortical bulging at 5-6 hours. Moderate bulging 8 hours. Loss of pulsations at 6 hours. Some diffusion of trypan blue into cortex at 8 hours |
| Experimental 5 | Adrenocorticotrophic hormone (ACTH) (ACTHAR-Armour) | 20 units/kg I.M. | 2 hours prior to performing operation. Booster dose 2 hours after operation | Moderate cortical bulging at 6 hours. Loss of cortical pulsations at 4 hours. Moderate diffusion of trypan blue into cortex at 6 hours |

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# ACUTE MONOCYTIC LEUKEMIA IN CHILDHOOD

MARTIN K. GORTEN, M.D. AND ROBERT HIDEY, M.D.†

Acute and subacute types of lymphocytic leukemia predominate blood dyscrasias in children (1). The remainder of childhood leukemias is composed mainly of the myelocytic and stem cell dyscrasias. This report is concerned with monocytic leukemia, also known as the histiomonocytic or Schilling type (2). The Naegeli or myelomonocytic type (2) of leukemia is a variant of the myeloblastic and myelocytic group of leukemias and is not considered here.

Monocytic leukemia has been established as a definite and separate entity and is frequently encountered in adults, but is unusual in the pediatric age group (1, 3). Reports of 49 isolated cases in childhood were found after a search of the literature (see Table 1). Some of these were listed as monocytic leukemias, but were not described. Other cases undoubtedly have remained unreported. It is apparent that monocytic leukemia has been observed with greater frequency since the advent of bone marrow examinations as a diagnostic procedure. The purpose of this report is to add another case of acute monocytic leukemia in childhood.

## CASE REPORT

This was the first and only University Hospital admission of this 29 month old, white male. The chief complaint was a persistent head cold, epistaxis and anorexia. The onset of his present illness dated back to 5½ months prior to admission. Since then he has had an almost constant head cold, at times running a temperature elevation from 100° to 102°F. He was given a total of 10 to 12 penicillin injections to combat this condition. The head colds were manifested by rhinitis, a sore throat, a dry cough sometimes productive of mucus, and an occasional epistaxis lasting from 5 to 30 minutes. Five months prior to admission, the mother became aware of a sallow complexion which was accompanied by increasing pallor. The parents noticed anorexia, easy fatiguability, increasing irritability and weakness, and some weight loss in conjunction with the appearance of pallor. Three months after the onset of symptoms, "easy bruising" was first observed which in the succeeding 2 month period increased in frequency and became more severe. He was lethargic and slept a great deal in the 2 week interval prior to admission. The day before entrance to the hospital he complained of severe pain in both legs.

The past history revealed an uneventful prenatal, natal and neonatal course. The dietary and developmental histories were well within normal limits. He received immunizations and vaccination between the sixth and eighth month of life. He suffered no diseases prior to his present illness.

The family history was non-contributory.

Physical examination revealed a pale, sleepy, well-developed, well-nourished, 29 month old, white boy who showed evidence of recent weight loss. He appeared chronically ill with a sallow hue to his skin. The child was fretful and uncooperative. His temperature was 101.6°F rectally. The head configuration and size were normal. The conjunctivae were very pale, and the sclerae were white. The pupils were round, regular and equal, reacting to light and accommodation. His extraocular movements were full. Fundoscopic examination revealed no abnormalities. Both ear drums were mildly injected and a healed perforation was present in the left drum. The mucous membranes of the nose, mouth and throat were pale. A moderate stomatitis was present. The gums appeared hyperplastic. His teeth were not carious. Both tonsils were enlarged and mildly injected. The neck

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TABLE 1

*Monocytic Leukemia in Childhood*

Cases reported in the literature

| YEAR | AUTHOR | NO. CASES REPORTED | YEAR | AUTHOR | NO. CASES REPORTED |
|---|---|---|---|---|---|
| 1921 | Rosenthal | 1 | 1936 | Klumpp (5) | 1 |
| 1928 | Hannema | 1 | 1936 | Kato (6) | 1 |
| 1929 | Muenzer | 1 | 1937 | Osgood (4) | 1 |
| 1930 | Dias | 1 | 1938 | Ernandez—quoted by Court (1) | 2 |
| 1930 | Lawrence | 1 | 1938 | Newns—quoted by Court (1) | 2 |
| 1932 | Mesropyan | 1 | 1939 | Court (1) | 2 |
| 1933 | Andreoli | 1 | 1939 | Booth (7) | 2 |
| 1933 | Cisi | 1 | 1944 | Blackfan & Diamond (8) | 1 |
| 1933 | Grenet | 3 | 1948 | Sullivan (9) | 1 |
| 1933 | Tidswell | 1 | 1949 | Dale (10) | 1 |
| 1933 | Gittins | 1 | 1950 | Moody (11) | 1 |
| 1933 | Levy | 1 | 1950 | Bierman (12) | 3 |
| 1933 | Foot | 1 | 1951 | Rodgers (13) | 6 |
| 1934 | Doan | 1 | 1951 | Wilson (14) | 6 |
| 1935 | Hawksley | 1 | 1952 | Legarreta (15) | 2 |

Preceding cases quoted by Osgood (4).
*Total number of cases reported* = 49.

was supple. Several pea-sized, discrete, non-tender lymph glands in both anterior and posterior cervical chains could be palpated. The trachea was in the midline. The thyroid gland could not be palpated. The chest appeared symmetrical, and the lungs were clear to percussion and auscultation. The respiratory rate was 20 per minute. The heart had a forceful apex beat and was mildly enlarged to percussion. The heart sounds were loud and harsh. Systolic mitral and pulmonic murmurs could be heard, but were not transmitted nor affected by a change in position. The pulse rate was 140 per minute. The blood pressure was 104 systolic and 68 diastolic in both upper extremities. The abdomen showed some venous tracery and was flat. There were no masses, tenderness, spasm or rigidity. The liver edge could be palpated to 3.5 cm. below the costal margin in the midclavicular line and was firm, smooth and non-tender. The spleen was readily palpated to 2 cm. below the costal margin and was hard, smooth and non-tender. There was no venous hum, and peristalsis was active. The skin showed many diffusely scattered petechiae, and 3 ecchymotic areas were observed on the lower extremities. The remainder of the examination was within normal limits.

### Laboratory Studies on Admission:

Hemogram: Hemoglobin 2.6 gm. per cent; erythrocytes 0.93 million/cmm.; MCV 89 cubic microns; MCH 29 micromicrograms; MCHC 33 per cent; reticulocytes 0.6 per cent; platelets 4,800/cmm.; leukocytes 10,600/cmm.; differential-PMN's 28 per cent; lymphocytes 30 per cent; monocytes 31 per cent; promonocytes 5 per cent; monoblasts 6 per cent. Urinalysis normal. Tuberculin skin tests were negative. Roentgenograms: All within normal deviation except for a heart that appeared round and slightly, but diffusely, enlarged, and the presence of hepato-splenomegaly. Bacteriology: Nose and throat culture revealed D. pneumoniae; blood cultures (3) no growth; agglutination tests negative; cold and heterophile agglutinations were within normal range. Biochemistry: Blood sugar, urea nitrogen, $CO_2$ combining power, chlorides, sodium, potassium, albumin, globulin, bilirubin, calcium, phosphorus, and alkaline phosphatase were all within normal range.
Bone Marrow (Iliac Crest) Aspiration:

Myeloid series: 1.6 per cent (neutrophils—1.3 per cent, eosinophils 0.3 per cent).

Lymphoid series: 17.4 per cent (blasts—2.7 per cent, young 0.7 per cent, mature 14.0 per cent)

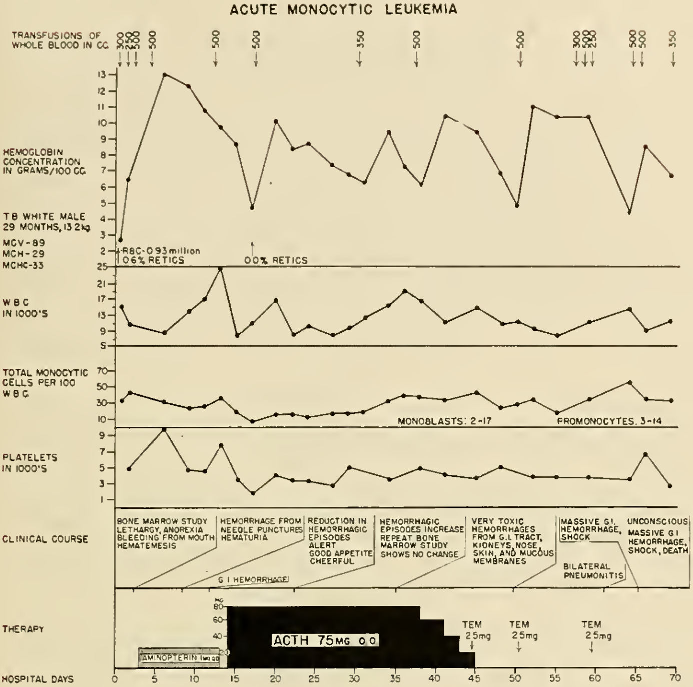

DIAGRAM 1

Monocytic series: 78.0 per cent (blasts—16.7 per cent, promonos 16.0 per cent, mature 45.3 per cent)
Erythroid series: 0.7 per cent (all normoblasts)
Primitive blast cells: 2.0 per cent.
Plasma cells: 0.3 per cent.

The marrow appeared hypercellular and was practically devoid of erythroid and myeloid elements. No megakaryocytes were seen. The marrow was almost entirely replaced by cells of the monocytic series, a number being quite immature.

**Course in the hospital:** (See Diagram No. 1)

The patient received 1 mgm. of Aminopterin® orally every day for 10 days. Because of a marked toxicity, an increase in bleeding episodes, and no change in blood aspects, the aminopterin was discontinued. The child was then started on a course of ACTH, receiving 75 mgm. every day. In spite of some subjective improvement and a transient decrease in the bleeding episodes, the patient's condition was deteriorating. ACTH therapy was discontinued after 31 days, and the child was started on triethylene melamine. He received 2.5 mgm. once a week without effect. Several antibiotics (penicillin, Aureomycin®, Chloromycetin®) were given successively for varying intervals in

TABLE 2

*Case of Monocytic Leukemia*

Serial Blood Counts

| Days in Hospital | Hemoglobin in gm. per 100 cc. | RBC in millions | Platelets per cmm. | WBC per cmm. | Differential Counts (in %) | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | PMN | Lymphs | Mono-cytes | Pro-mono-cytes | Mono-blasts |
| 1 | 2.6 | 0.93 | 4,800 | 10,600 | 28 | 30 | 31 | 5 | 6 |
| 6 | 13.0 | 4.85 | 9,800 | 8,850 | 22 | 48 | 13 | 10 | 7 |
| 9 | 12.2 | 4.66 | 4,600 | 13,900 | 26 | 52 | 8 | 8 | 6 |
| 11 | 10.7 | 4.59 | 4,500 | 17,050 | 25 | 50 | 15 | 6 | 4 |
| 13 | 9.6 | 3.96 | 7,800 | 25,700 | 15 | 53 | 27 | 3 | 2 |
| 15 | 8.6 | 3.39 | 3,300 | 7,550 | 36 | 45 | 8 | 9 | 2 |
| 20 | 10.0 | 4.03 | 4,000 | 16,700 | 30 | 50 | 13 | 3 | 4 |
| 22 | 8.3 | 3.28 | 3,200 | 7,950 | 23 | 60 | 12 | 3 | 2 |
| 24 | 8.7 | 3.24 | 3,200 | 10,350 | 15 | 71 | 8 | 3 | 3 |
| 27 | 7.3 | 2.84 | 2,800 | 7,800 | 16 | 66 | 10 | 6 | 2 |
| 29 | 6.9 | 2.59 | 5,000 | 9,650 | 13 | 68 | 13 | 3 | 3 |
| 31 | 6.3 | — | — | 12,650 | 8 | 71 | 10 | 8 | 3 |
| 34 | 9.4 | 3.52 | 3,500 | 15,200 | 26 | 43 | 24 | 4 | 3 |
| 36 | 7.2 | — | — | 18,900 | 25 | 36 | 29 | 5 | 5 |
| 38 | 6.1 | 2.40 | 4,800 | 16,450 | 24 | 38 | 33 | 3 | 2 |
| 41 | 10.4 | 4.05 | 4,000 | 11,050 | 29 | 40 | 16 | 8 | 7 |
| 45 | 9.3 | 3.65 | 3,600 | 14,550 | 32 | 28 | 15 | 14 | 11 |
| 48 | 6.8 | 2.92 | 5,200 | 10,800 | 22 | 57 | 11 | 3 | 7 |
| 50 | 4.9 | — | — | 11,100 | 25 | 50 | 18 | 3 | 4 |
| 52 | 11.0 | 3.90 | 3,900 | 9,350 | 27 | 41 | 22 | 5 | 5 |
| 55 | 10.4 | 3.95 | 3,900 | 8,000 | 19 | 60 | 14 | 3 | 4 |
| 59 | 10.4 | 3.74 | 3,700 | 11,050 | 14 | 55 | 19 | 5 | 7 |
| 64 | 4.4 | — | 3,400 | 14,150 | 24 | 23 | 26 | 10 | 17 |
| 66 | 8.5 | 3.42 | 6,800 | 9,000 | 30 | 37 | 17 | 10 | 6 |
| 69 | 6.6 | 2.55 | 2,500 | 11,150 | 31 | 36 | 13 | 8 | 12 |

addition to antileukemic therapy. The child received 500 mgm. ascorbic acid, twice a day, and 50 mgm. Rutin, daily. The serial blood counts are presented in Table 2.

Prior to the discontinuance of the ACTH, a repeat bone marrow study was performed which showed no appreciable change from the initial examination. The entire course was marked by many hemorrhagic episodes, persistent anemia and thrombopenia, requiring more and more blood transfusions. Transient improvement could not be observed at any time, and the patient's condition continued to weaken. On the 73d hospital day, following a massive gastrointestinal hemorrhage, the child expired. Autopsy permission was not granted by the parents.

## DISCUSSION

Monocytic leukemia differs but little clinically from other childhood leukemias (3). Certain minor differences exist and were described by Court (1). Cutaneous lesions (nodules or leukemic infiltration, boils, carbuncles and rashes) are observed in approximately 18 per cent of monocytic leukemias, but these are rare in other leukemias of childhood.

The diagnosis of monocytic leukemia can be made by hematologic as well as by histologic means. The peripheral blood and the bone marrow show a proliferation of monocytes of varying degrees of maturity. Some of the monocytes in the peripheral blood appear to be quite atypical, but these are the cells which are pathognomonic of the disease (6). More typical monoblasts, promonocytes and monocytes predominate in the bone marrow.

The course of the disease is usually progressive and rapid without remissions. The antileukemic agents have little or no effect in monocytic leukemia, an observation repeatedly made in adults. This is in contrast to acute and subacute lymphatic leukemias in children because a 50 to 60 per cent temporary remission rate is attained with the use of folic acid antagonists (14, 16) and with ACTH or cortisone (17, 18). Triethylene melamine, a compound with nitrogen mustard-like activity, has been used in the treatment of leukemias and lymphomas (19). Its efficacy in childhood leukemias has not been established. The patient in this report did not respond to any of the previously mentioned antileukemic agents. It might be added that the child needed numerous blood transfusions which seemed less effective than in other varieties of leukemia.

## SUMMARY

1. A case of acute monocytic leukemia in a 29 month old, white male child is reported with clinical and hematologic findings.

2. A review of the literature reveals this to be the fiftieth case reported in childhood.

3. Treatment with aminopterin, ACTH and triethylene melamine proved to be ineffective.

4. Monocytic leukemia constitutes a clinical and hematologic entity that is infrequently observed in the pediatric age group.

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# PANCREATODUODENECTOMY FOR CARCINOMA OF THE AMPULLA OF VATER*†

## Case Report and Review

C. REID EDWARDS, M.D. and ERWIN R. JENNINGS, M.D.

Radical pancreatoduodenectomy for the treatment of carcinoma in the region of the ampulla of Vater was revived by Whipple and co-workers (14) in 1935. Since that time modifications of the operation have been used in the treatment of carcinoma of the ampulla, pancreas, duodenum, and of the bile ducts. The results thus far have been inconclusive; and the 3 to 5 year survivals have been exceedingly few. However, the prognosis of carcinoma in this region is so poor by all other methods of treatment, it seems imperative to report any relative successes thus far obtained.

This is a case report of a patient with carcinoma of the ampulla of Vater on whom a pancreatoduodenectomy was done. Three and one-fourth years later a second operation was done because of a stricture at the old choledochojejunostomy stoma. During surgery no evidence was found of any lesion suggestive of carcinoma. However, the patient died approximately 6 months later with evidences of metastatic spread. Because of the fact that some relatively long range survivals have lived in the presence of rather extensive metastases, the opportunity of exploration 3 years postoperatively, with no gross evidence of a lesion, lends some support to the efficacy of pancreatoduodenectomy. Only time and evaluation of the lesions encountered will give the final answer to the question of proper treatment for lesions of this kind.

The incidence of carcinoma of the ampulla is relatively low. Lieber, Stewart, and Lund (8) included carcinoma of the lower bile ducts, terminal pancreatic duct, and peripapillary mucosa of the duodenum in their figures, and found the incidence of carcinoma to be 1.13 per cent of all carcinomas, or 0.09 per cent of all autopsies. These figures were computed from over 26,000 autopsies. Baggenstoss (1) found 0.21 per cent of carcinomas to be in the ampullary region from necropsies at the Mayo Clinic. Outerbridge (10) quotes Geiser's series of 5,865 cancer patient autopsies, in which 23, or 0.4 per cent, affected the duodenum. Approximately 70 per cent of cancers, occurring in the duodenum, are at the papilla. Even though cancer in the region of the ampulla of Vater is not commonly encountered, it is seen frequently enough to offer a challenge for a successful cure.

The age incidence in carcinoma of the ampulla parallels that of cancer of the gastrointestinal tract. Most cases are over 50 years of age. Baggenstoss (1) gives the average age as 56, and states that three-fourths of all cases are over 50. Lieber, et al (8) found the average age of males to be 53.4 years; and that of females to be 55.3 years. However, 13 cases, or 5.9 per cent of Lieber's collected series ranged from 15 to 35 years.

Males are more often affected than females in the ratio of approximately 3 to one. There seems to be no racial preponderance for this disease.

No definite signs or symptoms are pathognomonic of carcinoma of the ampullary region. The clinical aspect may be varied. The occurrence of jaundice, hepatomegaly,

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weight loss, palpable gall bladder, and blood in the stool should make one very suspicious of carcinoma, and exploration should be done as soon as feasible. Pain is present in almost 50 per cent of the cases. Vomiting and ascites are sometimes seen.

Pathologically, peripapillary carcinomas are usually small, firm, nodular masses, 1 to 3 centimeters in diameter. The 2 types seen are the papillary type and the ulcerating type. The papillomatous types sometime extend out into the duodenum, or may extend back up the pancreatic duct or the common bile duct. Because of the location of the lesion, symptoms usually appear early, and jaundice is quite common. Sharpe and Comfort (11) found 38 of 40 cases to have some obstruction to the common duct.

Extension locally by infiltration is frequently seen. The duodenal wall, pancreas, common bile duct, and contiguous structures are usually involved. Lymphatic spread is principally by peri-aortic, peri-portal, and peri-pancreatic nodes. The most common visceral metastases are to the liver by lymphatics or the portal vein. Peritoneal implants and pulmonary metastases are sometimes seen. Some think, that metastases are late. Outerbridge (10) lists metastases present in only 22 per cent of his cases, and in Baggenstoss' (1) series of 28 cases, 15 had no evidence of metastases at autopsy. However, the operability rate in most clinical series is less than 50 per cent; thus indicating a relatively high incidence of metastasis or spread.

No real relation can be drawn between carcinoma of the ampulla and cholelithiasis. Gall stones are seen in approximately 20 per cent of cases.

Microscopically, the tumors are usually adenocarcinomas. They consist of columnar or cuboidal cells arranged about lumina with variations from papillary structures to solid cords. Evidence of inflammation and necrosis is seen in the ulcerated type.

Benign neoplasms, sarcomas, and squamous cell carcinomas have been reported in this area.

The prognosis in untreated cases is very poor. Lieber, Stewart, and Lund (8) report 97 of 100 patients treated medically, and not subjected to surgery, died an average of 6.63 months after onset of symptoms. Outerbridge (10) mentions 47 medically treated cases whose average duration was 7 and one-third months between noticeable symptoms and death.

The surgical approach to the question of carcinoma in the region of the ampulla of Vater is directed toward palliation, local excision, or radical excision. In order to evaluate surgical results, the operative mortality, as well as the length of period of survival, must be considered.

Of Bartlett's (2) series of 28 cases treated by palliative procedures, there was a 25 per cent operative mortality and a survival average of 5 months. Gray and Sharpe (6) report a 39.1 per cent mortality and Orr (9) reports a 39.4 per cent mortality in purely palliative operations. Siler and Zinninger (12) report 4 cases, all of whom expired in less than one month. The results are thus uniformly poor.

Several approaches have been used for excising the primary tumor. Transduodenal excision, retroduodenal excision, and actual partial resection of the duodenum have been done. Brunschwig (3) treated 4 cases by means of transduodenal excision and 3 of these did exceedingly well. When reported, the shortest survival was 2 years and 3 months; and the longest survival was 4 years and 5 months. Hunt (7) collected most of the figures reported on this problem prior to 1941. Of 93 cases with transduodenal excision, there was a mortality rate of 29 per cent. There were 11 resections of the duodenum with a 45.4 per cent mortality and 5 retroduodenal excisions with a 40 per cent mortality. Hunt (7) also reported 15 pancreatoduodenectomies with a 26.6 per cent mortality. This series represented 124 cases of carcinoma of the region of the ampulla of Vater treated surgically. Of the 86 surviving cases in Hunt's series, 10 were living well over the third postoperative year. One of these survived 22 years.

In reviewing the reports on radical pancreatoduodenal resection, one is struck with the steady decrease in operative mortality. Brunschwig (4) gives a 26 per cent mortality in 23 cases, but reports 13 consecutive cases with but one death; a 7.7 per cent mortality rate. Whipple (15) quotes Parsons and Lockwood as having no deaths in 13 radical procedures; and Waugh as having only one death in 15 pancreatoduodenectomies. Waugh's previous operative mortality was listed as 26 per cent. Cattell (5) reports 56 pancreatoduodenectomies done for malignant disease with an operative mortality of 14.3 per cent; 20 of these cases were operated on for carcinoma of the ampulla of Vater with only one death, or a mortality of 5 per cent.

The results in terms of longevity are still not conclusive. However, increasing numbers of 3 year survivals are appearing in the literature, and an occasional 5 year survival is seen.

Waugh (13) reports the follow-up on 30 patients having pancreatoduodenal resection for malignant disease. Of 14 patients with carcinoma of the pancreas, only 3 were alive an average of 2 years following surgery. However, 5 of 8 cases who had carcinoma of the ampulla were alive an average of 2 years; and 3 of the 5 had lived between 30 and 36 months with no evidence of recurrence at that time. Cattell (5) lists a 30 per cent 3-year survival in his series. Of 12 patients with a 5 year follow-up by Cattell, 3 or 25 per cent were living 5 years or more, and 6 cases or 50 per cent, lived 3 years or more.

Whipple (15) points out that the radical approach to carcinoma of the ampulla is met with better results than for carcinoma of the pancreas. Taking results from 5 clinics, Whipple reports 8 patients who lived 5 years or more after removal of ampullary growths, 2 of them for over 7 years. Along with this, he speaks of five-year survivals for carcinoma of the pancreas in 3 patients, one of them for 9 years.

## *Case Report*

*Case #12033, C. E.*

A 54 year old white male salesman was admitted to the University Hospital on April 2, 1948 with the chief complaint of abdominal discomfort of 6 weeks' duration. The patient described his discomfort as a feeling of fullness principally in the mid-epigastrium and right lower quadrant. He had noticed that his stool had become light yellow and his urine dark. He estimated a 10 pound weight loss in the 10 days prior to admission. The past history was essentially negative, except for a cholecystectomy in 1941. An examination revealed a well developed white male of 54 years in no acute distress. His blood pressure was 114 systolic over 72 diastolic; temperature 99.2 F., pulse 64, respirations 20, height 5′ 11″ and weight 188 pounds. There was no evidence of cyanosis or icterus. The abdomen was moderately obese with the liver palpable 4 finger breadths beneath the costal margins. The liver was neither tender nor nodular. Hemoglobin 15.6 grams, white count 6,600, urea nitrogen 16, blood sugar 105, thymol turbidity negative, prothrombin time slightly elevated, albumin 4.60, globulin 1.43, Van den Berg direct 3.2, indirect 1.5. The urine was negative except for one plus albumin. A barium enema was negative, except for diverticulosis. A tentative diagnosis of hepatitis of unknown etiology was made and the patient was placed on bed rest, high carbohydrate, and high vitamin diet. He improved on this regimen and was discharged on April 8, 1948.

The patient was readmitted on May 25, 1948, because of painless jaundice of 5 days' duration. Since discharge, he had seemed to improve. However, his stool seemed to become lighter and his urine darker. Approximately 5 days prior to admission, he noticed that his skin was turning yellow. An examination revealed a markedly jaundiced white male in no discomfort. The liver was palpable 3 finger breadths beneath the costal margins, smooth, and not tender. This examination was essentially unchanged from the previous one, except for the icterus. His temperature was 99.2 F., pulse 100, respirations 22, blood pressure 158 systolic over 90 diastolic, urea nitrogen 18, Van den Berg direct 15, indirect 7.9; prothrombin normal, hemoglobin 112 per cent, white count 10,000 with a normal differential and platelets. His urine revealed 2 plus albumin and occasional casts. The urine was positive for bile. The stool was negative for bile and negative for blood (Benzidine). A decision was made to do an exploratory laparotomy.

During surgery the common bile duct was found to be approximately 2 centimeters in diameter. The bile duct was thin walled and tortuous. The head of the pancreas was palpated and a small mass about the size of an olive was felt in the second portion of the duodenum, and thought to be carcinoma of the pancreas. The liver was markedly enlarged, nutmeg in color and character, but with no evidence of cirrhosis. There were no enlarged lymph nodes in the region of the aorta, pylorus or hilum of the liver. The duodenum was mobilized without difficulty and retracted medially along with the head of the pancreas. A tumor was easily palpated in the region of the ampulla of Vater. The common bile duct was then divided between clamps and packed from the field. The greater and lesser curvatures of the stomach were freed up and the stomach divided between Payr clamps. The duodenum was freely mobilized and the third portion of the duodenum was divided transversely between clamps. The head of the pancreas was freed up with the duodenum, and the head of the pancreas divided by sharp dissection. Thus the pylorus and the first, second, and third portions of the duodenum, along with the head of the pancreas, were excised in toto. The distal end of the duodenum was then closed. An end to side gastrojejunostomy was done. Distal to this, a choledochojejunostomy end to side anastomosis was performed, and distal to the choledochojejunostomy, a pancreatojejunostomy was done. The operating time was 3 hours and 30 minutes. The patient was given 1,500 c.c. of blood during the operation and did well throughout the procedure.

Postoperatively, he had a period of oliguria and azotemia. However, urinary output finally returned to normal. The Van den Berg promptly fell to 3.4 direct and 0.84 indirect. The blood sugar stayed within normal limits. The patient was ambulatory on the third postoperative day. He was discharged, as improved, on the twentieth postoperative day, with a moderate amount of drainage persisting.

The pathology report described the gross specimen as a small, hard lesion at the ampulla of Vater, measuring 1.5 x 1 cm. The microscopic section from the pancreas showed normal structures, but there was a considerable amount of necrosis of the acinar tissue. The section from the ampulla showed a marked proliferation of the epithelium in papillary projections with glandular elements in the center of the projections. The papillary tissue was in a cystic space lined with cuboidal epithelium. At some sites, the epithelium showed marked malignant changes and took a sheet-like configuration.

*Impression:* Papillary Adenocarcinoma with mucoid changes in the ampulla of Vater.

The patient was again admitted on October 17, 1949, because of substernal pain. A diagnosis of coronary thrombosis was made, and this impression was confirmed by electrocardiogram. He was treated with bed rest, sedation, and dicumarol, and was discharged as improved on November 13, 1949.

The patient was re-admitted on July 11, 1951 because of anorexia, weight loss, and right upper quadrant discomfort of approximately 4 months' duration, with jaundice of approximately 3 weeks' duration. For almost 3 weeks, he had noticed a yellowish tint to the skin, with light stool, dark urine, and a weight loss of 25 pounds. An examination revealed a markedly icteric patient, in no acute distress. There was evidence of recent weight loss. The examination otherwise was essentially negative, except for the liver being palpable 6 finger breadths beneath the costal margins. Hemoglobin was 68 per cent, white count 17,100, prothrombin time 16 per cent, urea nitrogen 15, blood sugar 214, thymol turbidity 3.1, total protein 5.87 with normal A G ratio, Van den Berg direct 5.94, and indirect 3.6. A surgical exploration was done on July 13, 1951. Numerous adhesions were encountered on entering the abdomen. The gastrojejunostomy was easily located, the jejunum followed distally, and the junction of the jejunum and the common duct was identified. The common duct was found to be markedly distended, and after careful palpation, it was presumed that a stricture existed at the stoma. The duct was opened and approximately 500 c.c. of greenish stained thickened material resulted. Probes were introduced into the common duct, up into both hepatic ducts, and no obstruction was met. Exploration of the area revealed a stricture at the anastomotic site, but no evidence of malignancy or glandular enlargement. A choledochojejunostomy was done at the site of the opening in the distended common duct. Further exploration revealed no evidence of malignancy, the liver being large but otherwise unremarkable.

The patient ran an uneventful postoperative course. He was ambulatory on the second postoperative day and was discharged on his eighth postoperative day with a Van den Berg of 2.46 direct, and 1.98 indirect.

The next admission was on August 1, 1951, because of chills and fever. There was clinical evidence of a cholangitis. During his hospital stay, the patient responded well to the antibiotics, but developed another coronary infarction. He responded to routine treatment and was discharged as improved on September 4, 1951.

The final admission was on January 3, 1952. The patient had been asymptomatic until 4 days prior to admission, when he began to have symptoms of intestinal obstruction. He experienced severe vomiting and was admitted in a dehydrated uremic state. An examination revealed metastatic nodules within his abdominal scar. Despite intravenous fluid therapy and decompression, he became progressively worse and died on January 13, 1952. An autopsy was not granted.

## SUMMARY

1. A case report of a 3 years and 8 months survival, following pancreatoduodenectomy for carcinoma of the ampulla of Vater, is presented.

2. Several features of carcinoma in the ampullary region are briefly discussed.

3. Because of the decrease in operative mortality and the increase in the 3 to 5 year survivals, radical pancreatoduodenectomy would seem to be the treatment of choice for carcinoma of the ampulla of Vater.

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# BOOK REVIEWS

**Practical Therapeutics.** *By Martin Emil Rehfuss, M.D., F.A.C.P., Professor of Clinical Medicine and Sutherland M. Prevost Lecturer in Therapeutics, The Jefferson Medical College, Philadelphia, and Alison Howe Price, A.B., M.D., Associate Professor of Medicine, The Jefferson Medical College, Philadelphia. The Williams & Wilkins Company, Baltimore, 1951. Second Edition. 938 pages. Illustrated. Price $15.00.*

This rather voluminous text represents an attempt on the part of the authors, and their several contributors, to keep the busy practitioner as well as the medical student abreast of the ever changing methods that comprise the field of modern therapeutics. The book is an outgrowth of the course in general therapeutics conducted by the senior author at The Jefferson Medical College and as such is in its second edition, the first appearing in 1948.

At first glance, the manuscript appears too ambitious in scope, as the subject matter presented seems to deal with most therapeutic measures available to modern medicine. However, when considered as a basic reference for therapeutic procedures, which the busy physician can turn to for information as needed, the magnitude of material is desirable.

Basically, the book is well written by men of established reputation in the field of therapeutics and the subjects discussed are presented in a pleasing and readable outline form. The subject arrangement is that of four large divisions headed; General Therapeutic Principles, Symptomatic Therapy, Treatment of Specific Disorders and Special Treatment. The division dealing with General Therapeutic Principles is of special value since it contains sections devoted to prescription writing, dietary therapy and an interesting and informative bit on nursing problems of value to the physician. One of the better features of the division entitled Symptomatic Therapy is the collections of gems in therapeutic practicality included in a section headed Practical Points. The third major division is concerned with the treatment of specific disorders and discusses them by systems. The majority of common conditions are included with the unfortunate omission of those involving the nervous system. The Special Treatment division acts as coordinator for the preceding sections and is designed to include features that may have been omitted elsewhere. Disorders of the eye, ear, nose and throat and some aspects of atomic medicine are among the interesting subjects considered.

In any publication in which several contributing authors must deal with wide subject matter, differences of opinion as to the management of specific disorders may arise. In such instances in which the subject is frankly controversial, the authors have attempted to draw attention to the fact and have then presented the several available methods of therapy in order of preference.

The numerous tables, graphs and attractive as well as informative illustrations, enhance the value of *Practical Therapeutics* as a general reference and contribute to its recommendation as a worthwhile addition to the library of the physician.

*J. B. Workman, M.D.*

**Electroencephalography in Clinical Practice,** *Robert S. Schwab, M.D.,* W. B. Saunders Co., Philadelphia & London, 1951. 195 pages with 176 pages of Text, 10 pages of Glossary, 6 pages of Index, 106 Illustrations.

In this book Dr. Robert S. Schwab has presented a bird's eye view of electroencephalography. Its limitations and practical applications in the epileptic patient, in other neurological and neurosurgical diseases and in psychiatric disorders are discussed. Its practical use in research, with an unlimited frontier at present, is presented.

Though the purpose of this book is stated to be for neurologists, internists, psychiatrists and neurosurgeons, it would be of great value to the general practitioner and pediatrician who see so many epileptics, neurological and psychiatric patients initially, before they are referred to specialists. Knowledge of what he can expect of an electroencephalograph should be of great value to him in determining the consultation to be requested and thereby help him to be more of the family counselor in these conditions.

The last chapter dealing with laboratory organization, training of staff and interpretation of records can be used by all new laboratories and all old laboratories considering present and future changes.

The conciseness of the book, 176 pages of text, makes it a practical one for the busy physician to refer to for aid in interpreting reports.

Ruth L. Baldwin, M.D.

**Physical Diagnosis,** Fourth Edition. *Ralph H. Major, M.D.* 426 pages plus an index of 25 pages 469 illustrations. Published by W. B. Saunders & Co., in Philadelphia 1951. Price $6.50.

Excellent organization of subject material with great clarity of presentation mark this textbook as an ideal one for the student of Physical Diagnosis.

It well expresses its author's intention not to prepare an encyclopedia which will confuse or discourage the beginner. Yet it is amply inclusive to satisfy any practitioner who desires to refresh his diagnostic techniques.

Illustrations are supplied in profusion, and are of uniformly good quality; the printing is of the publisher's usual excellence, making for easy reading.

A brief chapter on techniques of history taking ends the book. It does not discredit the antecedent text, but could be omitted. This phase of the examination of the patient is of such importance, and is currently undergoing so much investigation and revision, that it is slighted by such necessarily superficial treatment.

Delightful little bits of medical history have so easily been written into Dr. Major's text that one hardly realizes that many of these original descriptions are centuries old, so timely do they seem. The reader, the medical practitioner or student, is treated to views of the original drawings of Laennec's stethoscope, and of the first clinical thermometer in use in 1646; observes original charts such as Biot's original tracing of the type of respiration to which his name is given; and reads for himself the original descriptions of eponymous diseases, signs and procedures. Such a techniques uniquely employed, enables this textbook to hold one's interest for chapter after chapter, and help, it earn its space on any physician's book shelf.

John B. DeHoff, M.D.

# CLINICO-PATHOLOGIC CONFERENCE

### From the Case Histories, University Hospital, Baltimore

### *Clinical History*

This patient, a 41 year old white male, who had borne the effects of rheumatic heart disease for at least 13 years, appeared at the hospital for relief of severe, burning epigastric pain. The pain, of one hour's duration arose in the midepigastrium and extended into the right lower abdominal quadrant.

One month before this admission the patient's heart failed. Edema and orthopnea developed. Many times, he had to sit upright and dangle his legs over the side of the bed to relieve his breathlessness. Less than 2 weeks later, use of the right leg was lost. The lost function returned after the patient arrived at the hospital, so he was not admitted. Sixteen more days passed and then sudden pain pierced his chest on the right side and in the back. This affliction lasted 4 days. A physician, believing pneumonia to be the cause, administered penicillin. After 1 day of this treatment, the patient, being seized by epigastric pain, nausea and vomiting, was admitted to the hospital.

The examiner at the hospital found cardiac hypertrophy, a systolic thrust, and a diastolic thrill. The heart action was irregular in rate, rhythm, and force. The difference between the apical and radial rates was 50 per minute. The apical impulse rate was 140 per minute. The heart sounds heard in the mitral area were distorted by diastolic murmur, split second sound, and gallop rhythm. Examination of the posterior aspect of the left chest disclosed diminished breath sounds, rales of fine quality, and dull percussion note. Tenderness and rebound tenderness were demonstrated over McBurney's point. Meanwhile, the systolic blood pressure was 126, and the diastolic, 74 mm. of mercury. The body temperature was 97 F. The respiratory rate was 30 per minute. The arterial pulsations in the feet were weak. The fourth digit of the left hand was missing.

The laboratory reported a leucocyte count of 15,600 per cubic mm. of blood. Ninety-eight per cent were neutrophilic polymorphonuclear granulocytes. The hemoglobin concentration was 26 per cent. Albumin, erythrocytes, casts and leucocytes were present in the urine. The stool contained blood.

The patient was tented in an oxygenated atmosphere. Terramycin, heparin, Etamon chloride and crystodigin were administered. In 4 days, the abdominal pain disappeared and the circulation improved in the right leg. Hematuria and albuminuria persisted. Bowel evacuation was frequent and productive of blood-containing fluid stools. Fifteen days after admission the patient was stricken by paresis of the left side. His speech was slurred. There were no pathologic reflexes. His psyche remained bright. Demerol was given prior to blocking the stellate ganglia. Shortly, abdominal pain recurred. The abdominal wall was rigid and tender. In several hours the abdomen was distended and peristaltic sounds ceased. Death occurred soon thereafter.

### *Clinical Discussion*

*Dr. Sidney Scherlis:* One wonders whether this patient was distressed by a complication of rheumatic heart disease or by an unrelated abdominal disease. It would be advantageous to know whether the nausea and vomiting followed the onset of pain or occurred concomitantly with it. Pain such as this man had might be caused by peptic ulcer, gallbladder disease, pancreatitis, appendicitis or small intestinal lesions. A peptic ulcer which perforates causes sudden, severe pain which is associated with abdominal rigidity and shock. In gallbladder disease the pain is sharp and constricting, and radiates to the scapular region. The pain in pancreatic disease is sudden and severe, and radiates to the back. In appendicitis, the pain is acute and precedes the nausea and vomiting by an appreciable time. If the source of pain is the small intestine, it is first experienced in the central abdominal area and then in the right lower quadrant. When rupture of an organ occurs, the pain follows the migration of the released fluid. If obstruction is present, vomiting may be an early sign.

Heart failure had a prominent role in this patient's illness. One might presume that incompensable strain or acute myocarditis was responsible.

The leg weakness or possibly paralysis is an important key to this illness, which could have been caused by a vascular occlusion in the leg or a cerebrovascular accident. In either case, one wonders what relationship might exist between the heart disease and the vascular mishaps. Mural thrombi or bacterial endocarditis could serve as sources of emboli.

The pulmonary abnormalities defined by physical examination are compatible with pneumonia or embolism. The latter is likely.

The diarrhea and melena could result from embolism to the superior mesenteric artery and paralytic ileus, gangrene, and peritonitis. The hematuria and albuminuria are probably the result of renal emboli.

Thus far my theory is that this patient, in addition to rheumatic heart disease, had either mural thrombosis or endocarditis, and embolism to the appendicular circulation, the brain and mesenteric artery. The presence of cardiac hypertrophy and auricular fibrillation could predispose one to embolic events.

*Dr. Charles N. Davidson:* This is a roentgenogram made on February 15, the date of admission. The right lung is clear. There is a localized, well circumscribed, dense area of infarction just above the diaphragm. The vascular pattern, because of congestion, is heavy. A very prominent pulmonary artery segment is seen. The left ventricle does not appear to be enlarged proportionately to the left auricular enlargement. On the right side the auricle is prominent. It is difficult to evaluate right ventricular enlargement from only a roentgenogram of the chest. The film of the abdomen made the same day does not reveal any striking findings. The kidneys are not well defined. The liver does not appear enlarged. The February 16 roentgenogram of the abdomen shows a marked, localized, distension of the small bowel. This film was made while the patient was supine so no fluid level is visible. More pulmonary congestion is seen on both sides in the film made February 24. A small amount of fluid has accumulated in the left side.

*Dr. Scherlis:* Roentgen rays help us a great deal. The first film of the chest did not show a grossly enlarged heart. This supports the theory that the patient had something acute involving the heart 1 month before admission, and as a result of treatment the heart became smaller. Roentgenography supports the diagnosis of multiple emboli to the lungs. I should expect the autopsy to disclose rheumatic heart disease with mitral stenosis; left auricular enlargement; acute rheumatic carditis superimposed upon a chronic lesion; multiple embolic phenomena involving the extremities, the lung and superior mesenteric artery with gangrene of the small bowel; and emboli to the brain.

*Senior Student:* Nothing was said about the RBC being found in the urine.

*Dr. Scherlis:* I mentioned that the patient might have had infarcts of the kidney.

*Senior Student:* It is stated that the patient was permitted to walk around the bedside. In view of the embolic phenomena, why was this allowed?

*Dr. Scherlis:* The presence of emboli would not keep the patient from being ambulative. If the emboli were in the heart, the heart would be pumping whether the patient is at rest or not. After digitalis the heart would beat more strongly and emboli would be more likely to break off.

*Senior Student:* Do you rule out subacute bacterial endocarditis?

*Dr. Scherlis:* Against that is the absence of anemia and lesions in the spleen. No cutaneous lesions were noted. No mention is made of blood cultures.

*Senior Student:* Did you say that the fact that he had no pathologic reflexes suggested embolism to the brain?

*Dr. Scherlis:* No. I said that because he had paralysis and aphasia I should suspect he had embolic phenomena involving the brain.

*Senior Student:* Do you think that abdominal surgery might have helped this patient?

*Dr. Hugh R. Spencer:* I think it would have hastened his death.

### *Pathologic Discussion*

*Dr. Hugh R. Spencer:* This patient had a long history of rheumatic heart disease. The basic lesions in the case are found in the cardiovascular system. The heart is large. It is dilated, and the wall of the left ventricle is hypertrophied. The weight is 700 grams. Grossly, this is not the type of heart seen in uncomplicated rheumatic mitral stenosis. The mitral valve leaflets are fibrotic and thickened. The mitral orifice is button-hole in shape. It will not admit the index finger. The cusps of the aortic valve are thickened and fused at their points of junction. The leaflets of the tricuspid valve are thickened and distorted, although the changes seen here are not as marked as those of the mitral valve. The chordae tendineae on each side are thickened and shortened. The endocardium of the left ventricle shows areas of scarring. Both auricles are dilated and each contains a thrombus which is attached to the endocardial lining.

Auricular mural thrombosis frequently occurs in dilated fibrillating auricles. The auricular thrombi in this case are the sources of the emboli which are found in both circulations. Emboli are found in the radicles of both pulmonary arteries and small hemorrhagic infarcts are seen in both lungs. There are emboli in the splenic artery and in the right renal artery. Approximately 90 per cent of the splenic substance is involved by multiple anemic infarcts. The right kidney contains multiple small cortical anemic infarcts. Another embolus is found in the ileo-colic artery and the terminal ileum. The cecum and ascending colon show hemorrhagic infarction. The peritoneal surfaces of these segments of the intestinal tract are dulled and the walls are thick and dark red in color.

This is a case of chronic rheumatic valvular disease. It is somewhat unusual in that all of the heart valves except the pulmonic are involved. Other residual rheumatic changes in this case are scarring of the wall of the left auricle and focal myocardial fibrosis. The auricular dilatation with fibrillation is responsible for the development of auricular thrombi and these in turn are the sources of the embolism.

While the changes in rheumatic fever are not limited to the heart, the principal effects of the disease are caused by the lesions in this organ. Rheumatic disease of the heart is commonly a pancarditis, i.e., endocarditis, valvulitis, focal lesions (Aschoff bodies) about the vessels in the myocardium, and serofibrinous pericarditis. The healing of these lesions brings about valvular fibrosis, frequently with calcification, myocardial fibrosis and residual pericardial fibrosis, with or without adhesions.

### *Anatomic Diagnosis*

Chronic rheumatic heart disease with involvement of mitral, aortic and tricuspid valves; focal myocardial fibrosis; cardiac hypertrophy and dilatation; auricular thrombosis, bilateral; embolism, pulmonary, splenic, ileo-colic and renal arteries with infarcts, lungs, spleen, terminal ileum, cecum, ascending colon and kidney.

## OBSTETRIC CASE REPORT*

R. K., a thirty-two year old, white, female para 0–0–0–0 had had a known mild hypertension for the past 16 years.

The present pregnancy was uneventful except for mild hypertension (average BP 140 systolic over 84 diastolic) and a mild attack of cystitis at twenty weeks. Labor began spontaneously at term (membranes ruptured spontaneously after about an hour of labor—the amniotic fluid being clear) and progressed normally for about 5 hours when the patient suddenly experienced constant abdominal pain and passed a large blood clot per vaginum. Fetal distress was evidenced by marked fluctuations in fetal heart rate from 160 to 100 to 80 per minute. Maternal blood pressure was 145 systolic over 95 diastolic and the pulse rate was 90.

### DISCUSSION

Since obstetric patients with pre-existing hypertension are prone to the development of both preeclampsia and abruptio placentae, this patient had been seen frequently during her prenatal course. Her total weight gain had been restricted to ten pounds. Now, the sudden appearance of excessive uterine pain and vaginal bleeding, together with evidence of fetal distress suggested the diagnosis of abruptio placentae.

### MANAGEMENT

Blood was immediately obtained for typing, cross matching, and a separate tube was observed for clotting which was normal. The clot remained firm. The latter procedure was performed to rule out afibrinogenemia which can be a serious complication of abruptio placentae. In order to determine the exact condition of the cervix as well as the position and station of the fetal head, the patient was examined without anesthesia under the "double set-up" technique in the operating room. The cervix was found to be 7 to 8 centimeters dilated and completely effaced, and the fetal head was 1 centimeter below the ischial spines. The consultant and operator were in accord that the quickest method of delivery would best solve this problem. It was decided to perform a hysterostomatomy (Dührssen's incisions) and to deliver the baby vaginally. A 500 cc whole citrated blood transfusion was started at once.

Under general anesthesia, incisions at "10 and at 2 o'clock" and about 5 centimeters long were made in the cervix which then completely retraced. Central episiotomy was performed. Mid forceps were applied to the fetal head with gentle intermittent traction. A 3714 gram apneic, full term, living baby was delivered. Minimal resuscitation was required and the baby was sent to the nursery in good condition. The placenta was delivered by the Brandt-Andrews method after 4 minutes, and showed about 1/5 of the maternal surface to be covered with adherent blood clots. The uterine cavity was manually explored to rule out rupture. The uterus contracted well. The cervical incisions were examined. No extensions were found, and the incisions were repaired. A laceration in the vaginal mucosa at eight o'clock, and the episiotomy were repaired. The subsequent course of both mother and baby was normal.

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* From The Department of Obstetrics, University of Maryland School of Medicine, Baltimore.

*Final diagnosis: Hypertensive cardiovascular disease. Premature separation of a normally implanted placenta during labor.*

This case illustrates the application of cervical incisions to facilitate delivery in the event of intrapartum hemorrhage and fetal distress. If the cervix had been dilated less than 6 centimeters and not completely effaced, or if relatively easy vaginal delivery had not been anticipated, abdominal delivery would probably have been the proper procedure.

If the obstetrician is familiar with the post-partum cervix through practicing routine cervical inspection immediately following delivery, the operation of hysterostomatomy is rather simple to do. Under proper technic, the incisions rarely extend and healing takes place nicely. The procedure is rarely indicated but in a few instances may be life saving for the baby and much simpler for the mother than abdominal delivery.

# MEDICAL SCHOOL SECTION

## DR. ARTHUR M. SHIPLEY HONORED

*Staff Library in University Hospital Named for Emeritus Professor*

A new and completely refurnished staff library has been erected in the connecting wing between the University Hospital and the Psychiatric Institute. It will be known as the Author M. Shipley Library.

The room, approximately 15 by 40 feet, is adequately supplied with shelving and rack space for journals including reading and work tables. The central portion of the room contains an oil portrait of Dr. Shipley presented by former residents, associates and friends. Dr. Shipley was guest of honor at a short informal luncheon and dedication held in the University Hospital on February 25, 1954.

Dr. Arthur M. Shipley reviews his portrait at dedication ceremonies for library named in his honor. Left to right—Dr. C. R. Edwards, Acting Professor of Surgery, Dr. T. B. Symons, Acting President of the University of Maryland, Dr. Arthur M. Shipley, Emeritus Professor of Surgery, Dr. H. Boyd Wylie, Dean, and Dr. Maurice C. Pincoffs, Professor of Medicine.

## DEPARTMENT OF PHARMACOLOGY

Dr. C. Jelleff Carr and Dr. Raymond M. Burgison will present scientific papers at the meeting of the Federation of American Societies for Experimental Biology to be held in Atlantic City.

Dr. John C. Krantz, Jr. will present a paper on the "Physiology and Pharmacology of the Autonomic Nervous System" before the American College of Physicians at a meeting to be held in Pittsburgh, Pennsylvania on May 18, 1954. Dr. Krantz will also speak at the meeting of the Barbour-Randolph-Tucker Medical Society of West Virginia on June 17, 1954. He will discuss "Recent Advances in Drug Therapy".

## THE THIRD ANNUAL PEDIATRIC SEMINAR

The Department of Pediatrics of the School of Medicine was the sponsor of a Pediatric Seminar held on Sunday, March 28, 1954. Speakers included Dr. Rachel Ash, Associate Professor of Pediatric Cardiology at the University of Pennsylvania School of Medicine who spoke on "The Management of Common Pediatric Cardiac Diseases". Dr. Sydney S. Gellis, Assistant Professor of Pediatrics at the Harvard University School of Medicine spoke on "Practical Aspects of Liver Diseases in Pediatrics". Dr. Edwin L. Kendig, Jr., Assistant Professor of Pediatrics at the Medical College of Virginia spoke on "Early Diagnosis and Treatment of Tuberculosis in Children". Dr. Marion B. Sulzberger, Professor and Chairman of the Department of Dermatology and Syphilology at the New York University Postgraduate Medical School spoke on "Some Dermatologic Problems Seen in Pediatric Practice". All Discussions were moderated by Drs. Harriet Guild and C. Loring Joslin. The Committee on Arrangements included Drs. Edward G. Field, Samuel Shipley Glick and Ruth W. Baldwin.

Mr. William (Bill) Lepson

## MR. WILLIAM (BILL) LEPSON RETIRES

Parking 384,000 cars is no small task particularly when the total accident rate is limited to 1 car and for a maximum damage of $29. This is part of the enviable record of Mr. William (Bill) Lepson who retired as Chief Officer on the University Faculty parking lot as of April 1, 1954.

Bill came with the University on January 21, 1936 and has been in charge of the parking lots since that time. Several years ago he was afforded the status of special state police officer. Bill has seen the parking problem grow, multiply and become very difficult. He has witnessed increase of University parking from a few cars to as many as 285. In recent years physicians have demanded excellent facilities and special services from Bill despite the growing lack of space. As a part of his record go many words of praise and compliments from those members of the staff who have seen the mounting difficulties besetting him. Many admire the efficient and straightforward manner in which he has assumed the tact necessary for the proper execution of his job and good relationship between himself and the members of the faculty. Bill is retiring from the University because he expects to operate a transfer company, partly in Baltimore and partly at his home in New Windsor, Maryland.

## SIXTH ANNUAL LUTHERAN HOSPITAL SYMPOSIUM

### *May 8, 1954 Session Sponsored by Medical Association of Lutheran Hospital*

The Sixth Annual Medical and Surgical Symposium at the Lutheran Hospital of Maryland under the auspices of the Medical Association of the Lutheran Hospital will be held on May 8, 1954 at the Nurses Home, 731 Ashburton Street in Baltimore.

The morning session will be devoted to a symposium on "Thin Bones, Old People, and Hormone Therapy" and will be conducted by Dr. Edward C. Reifenstein, Jr.

A luncheon will be served to all guests at 12 Noon.

The afternoon program will consist of a panel discussion on "Hypertension" with Dr. Louis Krause, Chief of Medicine, Lutheran Hospital and Professor of Clinical Medicine at the University of Maryland School of Medicine, presiding. Participants will be Dr. William A. Jeffers, Chief of the Hypertension Section, University of Pennsylvania School of Medicine, and Associate Professor of Medicine; Dr. Paul Kimmelstiel, Pathologist and Director of Clinical Laboratories, Charlotte Memorial Hospital, Charlotte, North Carolina; and Dr. Thomas J. O'Neill, Associate Professor of Thoracic Surgery, Graduate School, University of Pennsylvania and Associate in Thoracic Surgery Hahnemann Hospital, Philadelphia, Pennsylvania.

The symposium will be followed by the customary staff dinner beginning at 7 P.M.

## DR. BARNETT RESIGNS

### *To Accept Columbia Post*

Dr. Donald J. Barnett, recently Assistant Professor of Roentgenology at the School of Medicine has accepted a position in the Department of Roentgenology at the Presbyterian Hospital. This department is a part of the Columbia Presbyterian Medical Center, 168th Street in New York City.

## RESIGNATION

### *Dr. Kilby Leaves*

Dr. Walter L. Kilby, Professor of Roentgenology and successor to Dr. Henry J. Walton, Emeritus Professor of Roentgenology, resigned effective September 1, 1953. Dr. Kilby has entered the private practice of Roentgenology in the city of Baltimore.

## ITEMS

Dr. V. V. Brunst, until recently Research Associate of the Department of Anatomy, has accepted a position on the staff of the Rosell Park Memorial Laboratory in Buffalo, New York.

## DEPARTMENT OF DERMATOLOGY

The Department of Dermatology presented an exhibit on "Terramycin in the Treatment of Dermatoses, a Review of 1750 Cases" at the 1953 convention of the American Medical Association.

Dr. Harry M. Robinson, Sr. and Dr. R. C. V. Robinson presented a paper entitled "A Newer Treatment for Alopecia Areata" at the Southern Medical Association meeting held in Atlanta, Georgia in 1953.

Dr. Harry M. Robinson, Jr. was recently elected to membership in the American Dermatological Association.

At the December 1953 meeting of the Academy of Dermatology, Dr. Francis A. Ellis took part in a course entitled "Special Problems in Dermato-Histopathology". Dr. Ellis was also a consultant member of a round table conference on Histopathology. The department displayed an additional exhibit on "Benign and Malignant Dyskeratosis". Dr. Harry M. Robinson, Jr. presented a paper on "The Role of Antibiotics in the Treatment of Acne".

Investigative activities of the department include studies on Moniliasis as a complication of antibiotic treatment. Fungicides are currently being investigated by all members of the department as are the Antibiotics, Erythromycin and Magnamycin. Dr. Eugene Bereston and Dr. Harry M. Robinson, Jr. have been experimenting and observing the resulting characteristics of various fungi on a new media. The treatment of leg ulcers and further work on Scleroderma are also included in this current studies.

## DR. HUTCHINS DIES

Dr. Elliott H. Hutchins, surgeon and Professor of Surgery at the School of Medicine died on August 24, 1953, at the age of 73. An alumnus of the Johns Hopkins University School of Medicine, Dr. Hutchins was active on the staff of Mercy Hospital and on the Faculty of the School of Medicine.

## STUDENT WINS 1953 SCHERING AWARD

Mr. Irvin P. Pollack a member of the class of 1956 and a native of Baltimore was named the grand prize winner of the $500 cash Schering Award for 1953, presented by the Schering Corporation of Bloomfield, New Jersey.

Dean Wylie congratulates Mr. Pollack, grand prize winner of 1953 Schering Award.

An additional prize known as the "Dean's Award", for the best essay written by a medical student at the University of Maryland, was also presented at ceremonies held in Chemical Hall at the School of Medicine on Wednesday, February 3, 1954 at 4 P.M. Mr. Pollack's prize essay was entitled "New Concepts in the Treatment of Peptic Ulcer." The selection was based upon the ability of the student to explore current and recent research and to integrate the findings with rational practices in clinical medicine.

## MEDICAL LIBRARY NOTES

In the period from February through April 1954, the following donors gave books and journals to the library:

Dr. M. Paul Byerly
Dr. Frank W. Hachtel
Dr. Samuel M. Jacobson
Dr. Arthur M. Kraut
Dr. Helen Maginnis
Dr. Edwin J. Ward
Dr. H. Boyd Wylie

Mrs. Ruth Lee Briscoe presented the library with her framed retirement certificate which refers to her thirty-two years of loyal service to the Medical Library. This certificate has been placed in the Maryland Room with Mrs. Briscoe's picture and other important documents relating to her long career as medical librarian at the University of Maryland.

Library staff members of the University of Maryland Libraries of Medicine, Dentistry, Pharmacy, Nursing, and Psychiatry were hosts to the Baltimore Chapter of the Special Libraries Association on March 16. The meeting was held in the Psychiatric Institute Library and adjoining rooms with Dr. John C. Krantz, Jr., Professor of Pharmacology of the School of Medicine, speaker of the evening.

A recent acquisition now in constant demand in the library is Sobotta's *Atlas of Descriptive Human Anatomy*, translated from the German by Dr. Eduard Uhlenhuth, Professor of Anatomy at the University of Maryland School of Medicine. The third volume of the three-volume set has been published first, to be followed later this year by volumes one and two. Dr. Uhlenhuth's careful and scholarly translation for this American edition makes the work of added interest and significance to the library and its patrons.

While Dr. Uhlenhuth was working on the translation, a question arose which was of interest to but could not be answered by the medical library or by inquiries to other libraries. Was Dr. Sobotta still alive? No death notice or obituary could be found in library sources at the time. Dr. Uhlenhuth therefore sent a letter to Germany for information and received from Bonn a reply quoted here in part:

> Professor Sobotta was Director of the Anatomical Institute in Bonn from April 1, 1919 to April 1, 1935. He was made Professor Emeritus in 1935 as he had reached the age limit for active service . . . Professor Sobotta died the end of April 1945 in his 75th year of life. An obituary was not published since the few scientific journals did not accept obituaries because of the scarcity of paper prevailing at that time. And, at that time, the Russian authorities did not permit obituaries to be published in the "Anatomische Anzeiger" which was under Russian control.

Dr. Uhlenhuth's translation of Sobotta's text contains a biographical sketch of Sobotta in the preface.

## PHI LAMBDA KAPPA LECTURE

Dr. Reuben L. Kahn, Director of the Serology Laboratory of the University Hospital, Ann Arbor, Michigan, was the annual Phi Lambda Kappa Medical Fraternity lecturer speaking on "Tissue Response in Infection and Immunity". Dr. Kahn was presented by the XI Chapter and the Baltimore Alumni Club of the Phi Lambda Kappa Medical Fraternity on Friday, April 16, 1954, speaking in Chemical Hall.

## MARYLAND SOCIETY FOR MEDICAL RESEARCH NEWS

### *Baltimore Animal Aid Association Seeks Homes for Research Dogs*

Physicians residing in and about Baltimore who wish to adopt dogs used in medical research at the School of Medicine may obtain further information by means of a pamphlet describing the procedure of obtaining these animals. This pamphlet will give the story of the movement to place dogs in Maryland homes following their contribution to the research programs in the School of Medicine. All of the dogs thus obtained will be given a silver medal of the Baltimore Animal Aid Association and data concerning the operative procedure performed on them will probably be available. Information may be acquired by contacting the Maryland Society for Medical Research at Mulberry 5-5348.

The Maryland Society continues active with numerous lectures and film demonstrations before high school classes. Plans for the coming year include the continuation of hospital and medical school laboratory tours for high school students. The Society's film "Frontiers in Medical Research" will again be shown during the 1953–54 season. It is also planned to expand its film program.

### *Foes of Animal Experimentation Change Tactics*

There has recently been a change in the tactics of those opposed to animal experimentation. They no longer feature "horror stories" which depict the experimentor as a sadistic monster. The public appeal to this program has been lacking. They now profess a willingness to support medical research providing it is "cleaned up". A newly formed institute, "The Animal Welfare Institute" (A.W.I.) and the American Humane Association (A.H.A.) imply that all is not well in the medical laboratories. They also suggest that they are the ones best fitted to remedy the situation and are demanding legislation giving them the *powers of inspection and regulation.*

Their actual attitude is revealed by the stand they take on legislation requiring public pounds to turn over unclaimed and ownerless dogs to medical institutions. Invariably they bitterly oppose this solution to the animal procurement problem.

Physicians are cautioned against this new approach. The communities where proper laws safeguarding the procurement of laboratory animals have not yet been passed are being notified of the change in tactics of the former group known as anti-vivisectionists.

For further information concerning this change of tactics contact the local office of the Maryland Society for Medical Research, or write directly to Dr. Dietrich C. Smith, Secretary, 29 South Greene Street, Baltimore 1 Maryland.

## JOHN M. DENNIS HEADS DEPARTMENT OF RADIOLOGY

Dr. John M. Dennis, class of 1945, was recently named Professor of Radiology at the School of Medicine, replacing Dr. Walter Kilby who resigned to enter private practice.

Dr. Dennis is a graduate of the University of Maryland having received his Bachelor of Science degree in 1943. Following his graduation from the School of Medicine he served from 1945 to 1946 as rotating intern at the University Hospital. From 1946 until 1948 he served in the Army School of Roentgenology. After his service he became resident in radiology at the University Hospital until 1950. From 1950 to 1951 he was senior resident in radiology at the Hospital of the University of Pennsylvania. From 1951 until 1953 he was part time radiologist at the University Hospital and also was engaged in private practice. Aside from his training in ro-

Dr. John M. Dennis

entgenology, Dr. Dennis has had considerable experience in the use of radio active isotopes in medical practice.

## PUBLICATIONS OF THE FACULTY

### ALPHABETICAL LIST TO BE COMPILED ANNUALLY BY MEDICAL LIBRARY

Beginning in 1954, a list of publications by the Faculty of the School of Medicine for the preceding calendar year will appear in the Bulletin. This year a bibliography will be compiled by the staff of the Medical Library. The present list, covering the calendar year 1953, was compiled by Mrs. Florence R. Kirk, Reference Librarian

## STUDIES FROM THE SCHOOL OF MEDICINE, 1953

Adams, Thurston R. and Jennings, Erwin R.:

Diverticulitis of the colon. Bull. School Med. Univ. Maryland. **38:** 85, 1953.

Allen, Harry S., Jr., Beacham, Edmund G., and Keschner, Harold W.:

Hepatitis and hypokalemia in tuberculosis. Am. Rev. Tuberc. **68:** 136, 1953.

Baldwin, Ruth, Davens, Edward, and Harris, Virginia Goddard:

The epilepsy program in public health. Am. J. Pub. Health. **43:** 452, 1953.

Barnes, Thomas G. and Yeager, George H.:
Abdominal aortic aneurysm: Resection with preservation of the lumen. Surgery. **33:** 658, 1953.

Bereston, Eugene S.:
A survey of progress in dermatology. ACTH, cortisone, antibiotics and vitamins play an important role in dermatology. Clin. Med. **60:** 367, 1953.

Blair, Emil and Yeager, George H.:
Regitine®, clinical observations on a new adrenolytic and sympatholytic drug. Bull. School Med. Univ. Maryland. **38:** 20, 1953.

Brantigan, Otto C.:
Cervical sympathetic stimulation caused by carcinoma of the lung. Dis. Chest. **23:** 547, 1953.

Brunst, V. V., Sheremetieva-Brunst, E. A., and Figge, Frank H. J.:
A comparison of the reactions of the irradiated parts of the bodies of two day old mice and urodele amphibians to roentgen treatment. Am. J. Roengtenol. **70:** 283, 1953.

Brunst, V. V. and Clayton, R. J.:
Photographing small laboratory animals. Lab. Invest. **2:** 376, 1953.

Brunst, V. V. and Figge, Frank H. J.:
The influence of x-ray irradiation of a narrow band between the fore and hind limbs of mice of various ages upon their subsequent growth and development. Abstract. Anat. Rec. **115:** 290, 1953.

Brunst, V. V. and Figge, Frank H. J.:
The influence of roentgen rays upon the development of the mouse by local irradiation of some parts of the body. J. Lab. & Clin. Med. **41:** 534, 1953.

Cowley, R. Adams and Yeager, George H.:
Treatment of aneurysms with follow-up studies on dicetyl phosphate. Surgery. **34:** 1032, 1953.

Crosby, R. M. N.:
Treatment of post-pneumoencephalographic headache with B-pyridyl carbinol tartrate (roniacol tartrate). Am. J. M. Sc. **225:** 61, 1953.

Crosby, R. M. N. and Weiland, Glenn L.:
Xanthochromia of the cerebrospinal fluid. II. Preliminary description of several colored substances. Arch. Neurol. & Psychiat. **69:** 732, 1953.

Deane, Garret E., Furman, J. Earl, Bentz, Alice R., and Woodward, Theodore E.:
Treatment of meningitis with chloromycetin palmite®; results of therapy in twenty-three cases. Pediatrics. **11:** 368, 1953.

Deane, Garret E., Heldrich, Frederick J., Jr., and Bradley, J. Edmund:
The use of BAL in the treatment of acute lead encephalopathy. J. Pediat. **42:** 409, 1953.

Douglass, Louis H., Gilbert, Richard A., Kaltreider, D. Frank, and McNally, Hugh B.:
The evaluation of the Willet clamp scalp traction by means of the tokodynamometer and comments on the use of the clamp. Am. J. Obst. & Gynec. **65:** 40, 1953.

Douglass, Louis H. and Kaltreider, D. Frank:

Trial forceps. Am. J. Obst. & Gynec. **65:** 889, 1953.

Douglass, Louis H., Morrison, J. Huff, and Oster, Robert:

Cerebral dysrhythmia and acute toxemia of pregnancy. Obst. & Gynec. **1:** 287, 1953.

Durlacher, Stanley H., Freimuth, Henry C., and Swan, Henry E., Jr.:

Blood changes in man following death due to drowning. Arch. Path. **56:** 454, 1953.

Edwards, C. Reid and Jennings, Erwin R.:

Peptic ulcer. A five year review. Ann. Surg. **137:** 329, 1953.

Feldman, Maurice:

Multiple primary carcinomas of the small intestine: A review of the literature. Am. J. Digest. Dis. **20:** 1, 1953.

Feldman, Maurice and Myers, Philip:

Invagination of the pylorus into the base of the duodenal bulb caused by scirrhous carcinoma of the stomach. Rev. Gastroenterol. **20:** 109, 1953.

Feldman, Maurice:

The incidence of the coexistency of gastric and duodenal ulceration. Gastroenterology. **23:** 304, 1953.

Feldman, Maurice and Myers, Philip:

The diagnostic evaluation of erect positioning in cholecystography. Radiology. **60:** 222, 1953.

Feldman, Maurice and Myers, Philip:

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Visceral circulation. A Ciba Foundation Symposium. G. E. W. Wolstenholme, editor. Little, Brown publisher, Boston 1953. Book review. Science. **118:** 633, Nov. 20, 1953.

Ferguson, Frederick P., and Smith, Dietrich C. with the technical assistance of Brown, Frances C. and Hendrickson, Carolyn F.:

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Figge, Frank H. J.:

Cancer research, past, present, and future. Editorial. Bull. School Med. Univ. Maryland. **38:** 159, 1953.

Figge, Frank H. J. and Peck, George C.:

Cancer detection and therapy. Affinity of lymphatic tissues for hematoporphyrin. Abstract. Anat. Rec. **115:** 306, 1953.

Figge, Frank H. J. and Peck, George C., and McCafferty, Robert:

Affinity of lymphatic, neoplastic and other tissues for porphyrin compounds. J. Histochem. & Cytochem. **1:** 385, 1953.

Figge, Frank H. J. and Wichterman, Ralph:

The influence of hematoporphyrin, sodium nitrite, and nembutal on radiation sensitivity in paramecium. Proc. Am. Assoc. Cancer Res. **1:** 16, 1953.

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Managing the emotional problems of the cancer patient. CA, a bulletin of cancer progress. **3:** 19, 1953.

Gallaher, James P. and Douglass, Louis H.:
Sarcoidosis and pregnancy. Obst. & Gynec. **2:** 590, 1953.

Gilbert, C. Richard A., Valdares, Jose, Kaltreider, D. Frank, and Douglass, Louis:
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Hull, Harry C. and Ganey, Joseph B.:
The Henry approach to femoral hernia. Ann. Surg. **137:** 57, 1953.

Loewald, Hans W.:
Psychoanalysis and modern views on human existence and religious experience. J. Pastoral Care. **7:** Spring, 1953.

Lu, Go, Brahen, Leonard S., and Krantz, John C., Jr.:
Anesthesia. XXXVIII: Quantitative measurement of mucosal irritation of volatile anesthetics. Anesthesiology. **14:** 143, 1953.

Lu, Go and Krantz, John C., Jr.:
Anesthesia. XXXIX: The in vitro studies of the influence on adenosine triphosphate (ATP) dephosphorylation by central depressants and stimulants. Anesthesiology. **14:** 348, 1953.

Marriott, Henry J. L.:
Primary systemic amyloidosis. Editorial. Ann. Int. Med. **38:** 620, 1953.

Marriott, Henry J. L.:
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Miller, Joseph M., Ginsberg, Milton, and Scarborough, C. Parke:
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Morrison, Samuel, Ebeling, William Carl, and Bennett, Van B.:
A study of the reciprocal relationship of hypertension and gastro-intestinal bleeding. Maryland M. J. **2:** 328, 1953.

Morrison, Samuel and Feldman, Maurice:

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Newell, H. Whitman and Schultz, Kathryn L.:

Brief therapy for acute anxiety in a five year old girl. Workshop 1952. Am. J. Orthopsychiat. **23:** 186, 1953.

Peck, George C., Mack, H. Patterson, and Figge, Frank H. J.:

Study of the effect of hematoporphyrin on hypophyseal-adrenal relationships. Proc. Soc. Exper. Biol. & Med. **83:** 264, 1953.

Peck, George C., Mack, H. Patterson, and Figge, Frank H. J.:

Cancer detection and therapy. III. Affinity of lymphatic tissue for hematoporphyrin. Bull. School Med. Univ. Maryland. **38:** 124, 1953.

Powell, John W., Rayner, Jeannette, and Finesinger, Jacob E.:

Responses to disaster in American cultural groups. Symposium on Stress, Army Medical Service Graduate School, Washington, D. C. 1953, p. 174.

Powell, Perry O., Jr. and Savage, John E.:

Nisentil in obstetrics. Obst. & Gynec. **2:** 658, 1953.

Ramsey, J. H. and Reimann, D. L.:

Bronchial adenomas arising in mucous glands. Illustrative case. Am. J. Path. **29:** 339, 1953.

Reimann, D. L. and Mansberger, A. R., Jr.:

A technical consideration of cardiopneumopexy—comparative study. Bull. School Med. Univ. Maryland. **38:** 118, 1953.

Robinson, Harry M. and Robinson, Harry M., Jr.:

Broad-spectrum antibiotic therapy of early syphilis. Post-treatment survey. Am. J. Syph. **37:** 243, 1953.

Robinson, Harry M., Jr. and Cohen, Morris M.:

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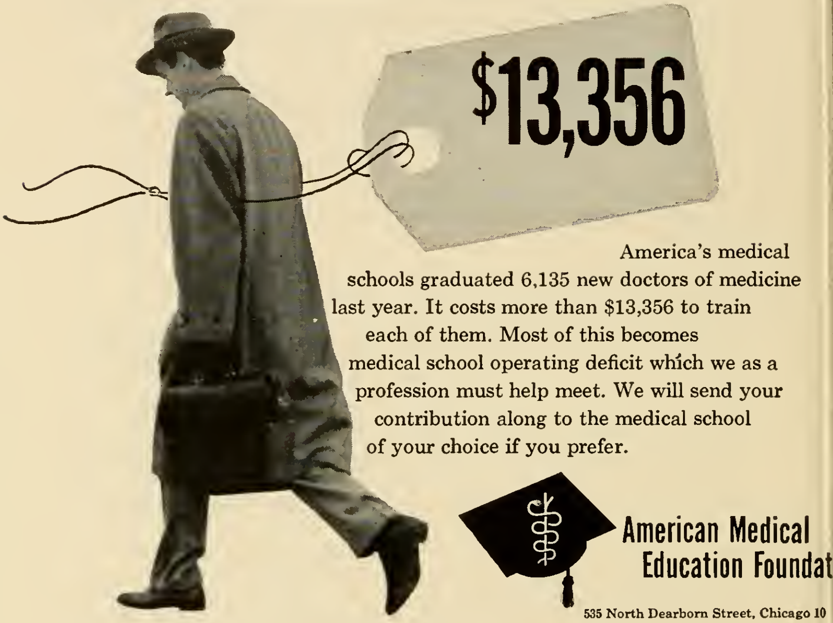

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# POST GRADUATE COMMITTEE SECTION

## POST GRADUATE COMMITTEE, SCHOOL OF MEDICINE

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### POSTGRADUATE COURSES—BALTIMORE CITY MEDICAL SOCIETY

Every week for the next 3 months, a series of lectures and clinics will continue to be presented by the Postgraduate Committee of the Baltimore City Medical Society in cooperation with the Maryland Academy of General Practice. The series was created this year by the Committee under the Chairmanship of Dr. Harry M. Robinson, Jr. The results have been most gratifying. The many physicians throughout the State who attended the first sessions on "Dermatology" were most enthusiastic.

A course on "Hematology" will begin on February the third, followed by a series on "Endocrinology" beginning March the fourth. The final course, "Disturbance of Water and Electrolyte Metabolism", will start on April the first.

The Postgraduate Committee of the School of Medicine, University of Maryland congratulates Dr. Robinson and all those with whom he is associated in this venture on its success.

### JOURNAL CLUB MEETINGS

A combined teaching program sponsored by the Departments of Gynecology and Obstetrics of the University Hospital was initiated November 4, 1953. These "Journal Club Meetings" are held the first Wednesday of each month at 7:30 P. M. in the Novey Memorial Room of the Hospital.

In attendance are members of the visiting staff of the Obstetrics and Gynecology Departments, the house staff of both services, and the residents in Medicine, Surgery, and Pediatrics of the University. Invitations have also been extended to the obstetric and gynecologic residents of several of the hospitals throughout the City.

At each meeting, the current literature is reviewed by house officers and discussed by both house and visiting staffs. The first program of 1954 was presented on January 6, by Dr. Louis H. Douglass. He discussed "Pelvic Tumors Complicating Pregnancy and Their Management". Dinner was served at 6:30 P.M. in the main dining room of the Hospital.

### EASTERN SHORE LECTURES

A series of lectures are being given at the Easton Memorial Hospital to a group of Eastern Shore physicians under the auspices of the Postgraduate Committee of the University of Maryland School of Medicine. The program of subjects to be discussed is as follows:

January 20, 1954 .............................. ALLERGY
Howard M. Bubert, M.D., Chief
Section of Allergy
School of Medicine University of Maryland

Jerome Sherman, M.D., Assistant Chief
Section of Allergy
School of Medicine University of Maryland
March 18, 1954 ...................................... SKIN
Harry M. Robinson, Jr., M.D., Chief
Dermatology and Syphilis Clinic
School of Medicine University of Maryland

R. C. V. Robinson, M.D.
Assistant Professor of Dermatology
School of Medicine University of Maryland

Israel Zeligman, M.D.
Assistant Professor of Dermatology
School of Medicine University of Maryland

William R. Bundick, M.D.
Associate in Dermatology
School of Medicine University of Maryland
May 19, 1954 .............................. PEDIATRICS
J. Edmund Bradley, M.D.
Professor of Pediatrics and Head of the Department
School of Medicine University of Maryland

## MEDICAL COLOR TELEVISION—STORY OF PROGRESS†

The Seventh Clinical Meeting of the American Medical Association, in St. Louis, will witness a milestone in the history of medical color television when the color television unit of Smith, Kline & French Laboratories presents its 50th program of colorcasting for the medical profession. Those familiar with the history of medical color television will remember that it was at the 1949 meeting of the A.M.A. in Atlantic City where this firm's TV unit presented the first program of operations and clinics to be televised in color. Since then it has carried its services to 15 states, Canada, and France. Many "firsts" were recorded along the way. In 1951, the Canadian Medical Association was host at the first program of color television seen in Canada. From here, the unit went abroad to give Europe its first look at color TV in a program presented at the International Society of Surgeons meeting in Paris.

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† Reprinted with permission from The Journal of the American Medical Association, November 21, 1953, page 1100.

In the first transcontinental color transmission, a coarctation was televised at the Los Angeles County Hospital and received in Chicago and New York.

Innovations in production and equipment have been introduced at almost every meeting, though many of these refinements are not obvious to the program viewers. The annual meeting of the A.M.A. in New York last summer saw the unveiling of what might be considered—from the viewpoint of the audience—the most striking additions to SKF equipment in years. The telecasts there were received on two 4½ × 6 foot screens, specially built by the Columbia Broadcasting System. These huge screens provide a picture 27 times larger than that seen on the 12-inch receivers used previously, rendering an image of great clarity. When the camera is focused on a surgical field, the view obtained by physicians in the audience is often better than that available to some members of the operating team. The dramatic character that the screens lend to surgical procedures was pointed out at a recent medical meeting when a mitral commissurotomy was being televised. The exposed heart filled almost the entire screen, and it was noticed that some persons in the audience were unconsciously moving their heads in time with the heartbeats. Another important addition to the equipment was the acquisition of a studio camera in 1951. Originally, the telecasting was done with but one surgical camera, employed for clinical presentations as well as operations. The physician conducting a clinical discussion was forced to adapt his presentation to the narrow confines of a camera designed primarily for use in an operating room. With the addition of the versatile studio camera, the variety of material used in clinical presentations has been greatly increased.

The production end of the telecasts has also undergone considerable change and improvement. Much of the responsibility for this progress can be attributed to the physicians participating in the programs. Whenever possible, they have utilized visual material in the preparation of their clinics. Through the development of production techniques, several very effective methods of presenting operations are now available to the surgeon. He can do a straight running narration as he operates, carry on a question and answer session with a member of his operating team, hold a round-table discussion with other surgeons watching the operation on a monitor located in the studio, or periodically switch to an assistant in the studio who can discuss and demonstrate clinical material pertinent to the surgery.

Perhaps the greatest change of all is to be found in the motives of the persons attending the program—the audience itself. For the first few years after the initiation of color television, almost every medical meeting the SKF unit visited was seeing color television for the first time. Naturally, the sheer novelty of the medium was a strong factor in inducing the doctors to attend the telecasts. It was difficult to ascertain whether the intended purpose of the medium—that of supplementing the postgraduate teaching programs of the meetings—was successful. As it developed, the audiences did not get smaller when the element of novelty faded; rather they increased in size. This was gratifying testimony to the place medical color television had won in the estimation of physicians no longer impressed with its "newness".

One might ask just what advantages color television brings to medical meetings. Without going into detail, a few of color TV's contributions to medical education can be cited. Only through the eye of the camera can hundreds of persons be brought to within a few feet—or a few inches—of interesting visual material. In a matter of seconds, the camera can accommodate itself to objects as different in size as a minute lesion and an iron lung. On the lecture platform, it is often inconvenient for speakers to introduce patients or even other specialists into their presentations. With the TV camera, such introductions are easily handled; in fact, they are highly desirable. The doctor-participant can also illustrate graphically the points he wishes emphasized. This visual reinforcement gives presentations much more impact than mere verbal references to points. Television can shift scenes readily. Thus an orthopedist can be televised giving a clinical demonstration of osteogenic sarcoma and, a few minutes later, the same surgeon can be shown performing a resection and bone graft to alleviate this condition in a patient. Unlike films, the presentations are individualized and completely natural. Because they are unrehearsed, the programs in general and the operations in particular are subject to all the drama of the unexpected. If a surgeon encounters unforeseen difficulties, the picture is not blacked out and the hundreds of persons in the audience are given the privilege of watching a surgeon cope with an immediate problem. Finally, there is color. The sharp contrast between color and black-and-white in the demonstration of medical subject matter will be seen by physicians attending the St. Louis meeting. The engineers will, from time to time, switch the camera to black-and-white. The loss of definition, depth, and detail will be obvious, as will be the change in the richness of the picture. The all-important asset of color, in combination with the advantages already mentioned, gives color television its surpassing distinction as an instrument for medical teaching.

# ALUMNI ASSOCIATION SECTION

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The names listed above are officers for the term beginning July 1, 1953 and ending June 30, 1954.

## PRESIDENT'S LETTER

As a member of the faculty of the School of Medicine it occurred to me that some comment on the activities here would be of interest to those of you in far places. It should be understood that any opinions expressed, are those of one individual and may not be entirely in accord with the authorities whose duty it is to guide the destiny of the school.

Shortly after the close of World War II it was evident to everyone that conditions here were such that it would not be possible for us to continue to function as a Grade A school. A "Planning Committee" was formed and did a prodigious job in bringing to light our deficiencies and making recommendations for their correction. A full report was compiled and presented to the President and the Board of Regents. This was accepted and many of the recommendations acted upon at once. In other instances time was necessary before definitive action could be taken.

Just recently it was announced that a Director of Medical Education and Research, had been appointed. (An official notice of this and other recent appointments will be found elsewhere in The Bulletin and it is of this position that I wish to comment.)

As it seems to be understood, this position in essence is that of a representative of the President of the University, located in Baltimore, and charged with the responsibility of medical education in all of its aspects. This will include the School of Nursing, the University Hospital and the School of Medicine. He will have the authority to make all decisions which would ordinarily be made by the President and to formulate policies relevant to medical education and research.

The filling of this position should be of immense value to the medical school, the hospital and the nursing school. To have on the scene an individual interested in medical education, who can see for himself our needs and who has the authority to fulfill these needs can result in a great improvement generally. There are two questions to be answered. One: Has the right man been appointed, and two: will he actually be given the authority necessary to do a good job? These questions can only be answered after sufficient time has elapsed. From all information available at present it would appear that the first question can be answered in the affirmative. As to the second, we can only hope and trust that the answer to that will also be yes. The potentialities are present and the Faculty will cooperate fully.

Two new departments have been established, Neurology and Preventive Medicine and Rehabilitation. There was a great need for these departments, and the appointees to each have been excellent choices. It is safe to say that much will be heard from these two departments in the near future.

The Departments of Roentgenology and Anesthesiology are now on a full-time basis. This is as it should be in a large medical center where the first consideration is teaching. Here again it is the opinion of this observer that the choices are excellent and that it would be very difficult to improve upon them. Already results are evident and there should be many more advancements in the near future.

With the creation of the Department of Preventive Medicine and Rehabilitation, there occurred a vacancy in the Faculty, the Professorship of Medicine. Following the general policy this position was filled by the appointment of a comparatively young man, one who has already proved his worth, and who will grow with the department. As with the other appointments, those that made the selection are to be congratulated upon their wisdom and astuteness.

The trend toward full-time clinical departments which began some 5 years ago has continued as vacancies have occurred. It is expected that in a few years all of these departments will be headed by individuals who will devote their entire time to teaching, research and administration. One of the most encouraging aspects of the plan as it is carried out here is the financial arrangements. These men receive their salary from the school and are not allowed to have private patients from whom fees are collected. In some schools fees from private patients make up a very valuable part of the income of the institution in that they exceed the salary paid the incumbent. This places the school in the position of engaging in the practice of medicine and puts it into unfair competition with the physician in private practice. That this is not true of our own school is a source of great satisfaction to everyone on the Faculty. It should help to create much better relations with the medical profession of the state.

In the fairly near future several other changes in personnel are to be made and it would appear that each new appointment will be most carefully considered. In addition the "Planning Committee" has been re-established and it is expected that there will be several changes in the curriculum and in the overall policies of the school. Those which have already been put into effect lead definitely to better medical educations and to more qualified physicians. Since everyone on the Faculty is vitally interested in this objective, we should continue for many years to be proud of the fact that we are graduates of the School of Medicine, University of Maryland.

Louis H. Douglass, M.D.
*President*

## ITEMS

**Dr. Zack D. Owens,** class of 1930, of Elizabeth City, North Carolina, was recently installed as President of the Medical Society of the State of North Carolina.

**Dr. Stanley N. Yaffe,** class of 1944, has announced the practice of Dermatology and Syphilology at the Latrobe Building, 2 East Read Street in Baltimore.

**Dr. Joseph G. Bird,** class of 1942, formerly a member of the Department of Pharmacology at the School of Medicine and an Associate Editor of the Bulletin of the School of Medicine, has recently accepted a position with the Sterling-Winthrop Research Institute in Rensselaer, New York. Dr. Bird will conduct initial administration of newly developed drugs to patients. This testing will be done in the Massachusetts General Hospital, in the Sloan-Kettering Institute and at other approved hospitals throughout the nation.

**Dr. N. Edward Nachlas,** class of 1947, has announced the opening of his office in the Latrobe Building, Charles and Read Streets, Baltimore 2, Maryland for the practice of otolaryngology, rhinoplasty and otoplasty.

**Dr. Edwin L. Seigman,** class of 1941, is currently stationed at the United States Naval Hospital in St. Albans, New York.

**Dr. Joseph J. Bowen, Jr.,** class of 1941, has announced the transfer of his office to the Lilley Building, 111 West Main Street, Waterbury 2, Connecticut. Dr. Bowen limits his practice to internal medicine and cardiology.

**Dr. Perry Futterman,** class of 1944, has been recently recalled to active duty with the United States Navy.

## ALUMNI TO GATHER AT SOUTHERN MEDICAL MEETING

Advance notice is given to the effect that the Medical Alumni Association will organize a dinner some time during the annual meeting of the Southern Medical Association which will be held in St. Louis, Missouri on November 8–11, 1954.

A subsequent edition of the Bulletin will carry additional details. Of course, if you attend the convention, notice will be posted concerning the University of Maryland dinner near the registration area.

# FRATERNAL NEWS

## PHI DELTA EPSILON

In 1953, Phi Delta Epsilon celebrated its 50th Anniversary as one of the nation's largest and most outstanding medical fraternities. Founded in 1904 at the Cornell University by a group of 8 young men, the fraternity has increased its ranks to include membership of over 10,000 physicians and students.

The Baltimore Chapter was originally founded as the Delta Chapter at the Baltimore Medical College in 1906. The second chapter in the district was the Epsilon Chapter at the University of Maryland. Iota Chapter at the College of Physicians and Surgeons in Baltimore was next chartered. Lambda Chapter at the Johns Hopkins University was chartered in 1911. In 1912 Delta and Epsilon Chapters merged through the amalgamation of the Baltimore Medical College and the University of Maryland taking on the name of Delta Epsilon at the University of Maryland. The chapter at the College of Physicians and Surgeons continued until 1915.

Delta Epsilon Chapter has been proud of its attainments in the high standards of ethics, scientific achievement and educational progress. The annual Phi Delta Epsilon Lectureship brings to the School of Medicine some of the outstanding and internationally known scientists. These have included Drs. Walter C. Alvarez, Homer W. Smith, Paul C. Aebersold, and Hans Selye. Each month the undergraduate chapter invites some outstanding local physician for an evening of instruction and informal discussion. A clinico-pathologic conference is held after each business meeting.

Phi Delta Epsilon graduates are active on the teaching staff of the School of Medicine. In particular, Dr. Samuel Shipley Glick, Assistant Professor of Pediatrics, has been honored by nomination to membership on the National Grand Council of Phi Delta Epsilon. Other Phi Delta Epsilon alumni who are on the School of Medicine staff include the following.

Dr. Charles Bagley, Jr.—Emeritus Professor of Neurosurgery

Dr. Louis V. Blum—Associate in Medicine. Dr. Blum is former President of the Baltimore Graduate Club.

Dr. Lester H. Caplan—Instructor in Pediatrics. Dr. Caplan is at present Vice-President of the Baltimore Graduate Club and is Treasurer for the 1953 Convention.

Dr. Morris M. Cohen—Instructor in Dermatology

Dr. Herbert Copeland—Instructor in Roentgenology

Dr. Daniel Ehrlich—Assistant in Obstetrics. Dr. Ehrlich has recently been recalled into the Army.

Dr. Jerome Fineman—Assistant Professor of Pediatrics

Dr. A. H. Finkelstein—Associate Professor of Pediatrics

Dr. Irving Freeman—Associate in Medicine. Dr. Freeman is President of the Baltimore Graduate Club.

Dr. Moses Gellman—Associate Professor of Orthopedic Surgery

Dr. Albert E. Goldstein—Assistant Professor of Pathology. Dr. Goldstein is Chief of the Hoffberger Research Laboratory at Sinai Hospital.

Dr. H. L. Granoff—Instructor in Gynecology

Dr. Isaac Gutman—Instructor in Orthopedic Surgery

Dr. Alvin Hartz—Associate in Medicine

Dr. Mark Hollander—Instructor in Dermatology and Syphilology

Dr. Marvin Jaffe—Fellow in Psychiatry

Dr. Joseph I. Kemler—Associate in Ophthalmology

Dr. Arnold Lavenstein—Instructor in Pediatrics

Dr. H. E. Levin—Associate in Bacteriology

Dr. Ephraim T. Lisansky—Associate Professor of Medicine; Associate in Psychiatry

Dr. I. H. Maseritz—Associate in Orthopedic Surgery

Dr. John Rosin—Instructor in Proctology

Dr. Leonard Scherlis—Assistant Professor of Medicine; Associate in Pediatrics

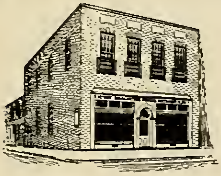
Dr. Sidney Scherlis—Assistant Professor of Medicine; Associate in Pediatrics

Dr. Albert Shapiro—Assistant Professor of Dermatology. Dr. Shapiro is a former President of the Baltimore Graduate Club.

Dr. I. A. Siegel—Associate Professor of Obstetrics. Dr. Siegel is Chief of Obstetrics at Sinai Hospital. He is a former District Deputy Grand Consul of the Washington, D. C., Maryland and Virginia District of the fraternity.

Dr. Herbert L. Warres—Instructor in Roentgenology.

Baltimore was host to the fraternity's Forty-ninth Annual Convention which was held at the Lord Baltimore Hotel on December 26 and 27, 1953. Dr. Bernard Kleiman was General Chairman of the Convention. Over 300 physicians and students were present.



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*Bulletin* OF
THE SCHOOL OF MEDICINE
*UNIVERSITY OF MARYLAND*

VOLUME 39 *July, 1954* NUMBER 3

# RELOCATION OF THE DIAPHRAGM TO OBLITERATE DEAD SPACE FOLLOWING PULMONARY RESECTION*

FRANK A. FARAINO, M.D. AND OTTO C. BRANTIGAN, M.D.
BALTIMORE, MARYLAND

Ever since the advent of pulmonary surgery thoracic surgeons have been confronted with the problem of dead space in the thoracic cavity. Following excisional therapy the restitutional compensatory mechanism is elaborated into various forms.[1] On the operative side the volume is decreased in all three dimensions: the vertical, transverse, and anterio-posterior; the diaphragm elevates, the height of the dome of the thoracic cage lowers through disuse, the interspaces become narrower, scoliosis develops toward the operative side and the mediastinum shifts in that direction. There is also an outpouring of serosanguineous fluid to help diminish the volume of the empty space. One of the major space-filling mechanisms concerns the residual lung tissue, which responds by increasing its volume.

The anatomic status of the remaining lung tissue is still not a settled point. Bremer[2], working with animals in their growing state, showed that a real hyperplasia develops in the remaining tissue; adult animals did not show regeneration or emphysema but simple distention. Similarly, Rienhoff, Reichert, and Heuer[3], and Behrend and Mann[4], were unable to find any emphysema but only simple distention. The same results were recorded by Rienhoff[1] in reporting on 12 cases of pneumonectomy performed on adult human beings.

On the other hand, the development of a true emphysema in Virchow's sense of the word has been demonstrated by various workers. Phillips et al[5], and Longacre and Johansman[6] reported "the breaking of the alveolar walls, thickening and clubbing of their broken ends, collapse of capillaries and possible fragmentation of elastic tissue" after resection in dogs. Paine[7] in 1940, was able to consistently produce emphysema in dogs by increasing the capacity of the chest.

Functionally, the deleterious effect of emphysema is accepted. The question arises whether overdistention is physiologically harmful in the early and in the late postoperative periods and whether ultimately it will develop into emphysema. Here again, when applied to the human subject, the results are not yet clear. Longacre, Carter, and Quill[8] showed a definite reduction of function in adult animals following pneumonectomy. Lester, Cournand, and Riley[9], in discussing pulmonary function in pneumonectomized children, demonstrated a constant reduction in breathing reserves although a good adaptation resulted. Cournand and Berry[10], in a report on cardiopulmonary function in 12 adult patients, showed a "reduction in breathing reserve in various states of activity," this being dependent upon the overdistended state of the remaining lung. A subsequent thoracoplasty was of decided advantage in relieving the detrimental effect caused by the overdistention. Birath[11], in a long term study of 12 postpneumonectomy and 14 postlobectomy patients believed that should emphysema develop in later years, "it appears generally to remain within normal limits and not to encroach upon function to any degree worth nentioning".

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* From the Department University of Mar of Thoracic Surgery,yland School of Medicine.

While the question of whether overdistention develops into emphysema is being established, thoracic surgeons have attempted to impede or prevent the formation of overdistention by various means.

Following pulmonary resection the generally accepted methods of treating the empty space fall into three categories. In the case of pneumonectomies, a serosanguineous exudate fills the pleural space. Later this clots and with subsequent fibrosis the mediastinum is pulled toward the resected side. Enlargement of the remaining lung results. In pulmonary resections of lesser caliber, the residual lung tissue is allowed to overdistend to fill the empty space.

A second method of managing the empty space utilizes a prosthesis. Space-filling material of various types have been used. Oils, paraffin, and metals such as stainless steel, tantalum, and zirconium, and numerous kinds of plastics[12] have been tried. All have the disadvantage of a harmful and often serious foreign body reaction.

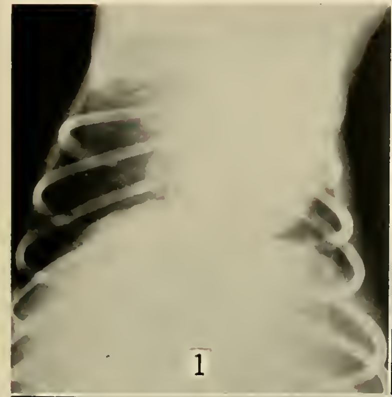
The most widely used procedure is the performance of a thoracoplasty, postpneumonectomy or postlobectomy, concomitantly or as a secondary procedure. This method gained impetus as a result of the physiologic studies of Cournand and Birath[10, 11] and their coworkers. Cournand and Berry[10], in a discussion of the physiologic effects after pneumonectomy, reported that in some patients a supplemental thoracoplasty was of distinct benefit. They found that in both children and adults the exercise tolerance was greater in those patients in whom overdistention was prevented by thoracoplasty.[9, 10]

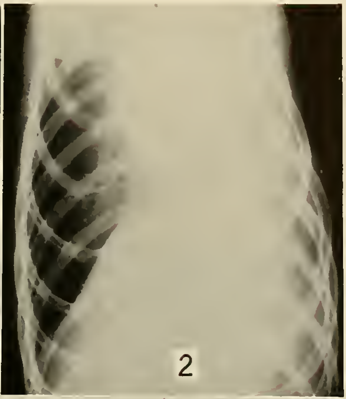
However, selective or radical thoracoplasty is a formidable procedure fraught with the risk of increasing mortality and morbidity rates. An alternative method is here presented to diminish and obliterate the dead space without the attendant dangers of previous procedures. It is proposed that the diaphragm be detached and relocated to a higher position.

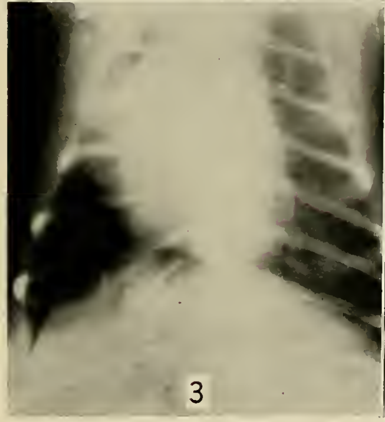
A single reference of relocation of the diaphragm has been found in the literature, this by Henschen[13], who in 1914 used this procedure for collapse therapy in dogs.

## MATERIAL AND METHOD

Six control and 6 experimental adult mongrel dogs have been used in this preliminary procedure. Right and left upper and lower lobectomies and pneumonectomies were performed on the control group; In addition to these procedures a relocation of the diaphragm to various levels was carried out on the experimental animals.







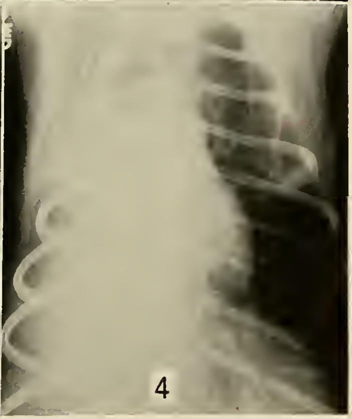


Fig. 1. Postoperative roentgenogram of control dog 17 illustrating shift of mediastinum toward operative side.

Fig. 2. Postoperative roentgenogram of experimental dog 25 showing mediastinum in midline

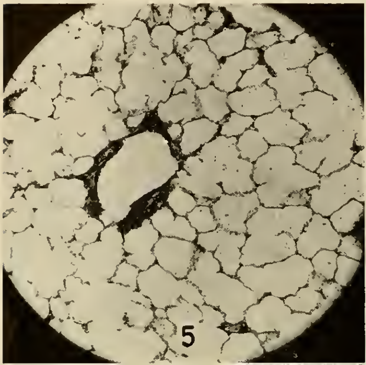
Fig. 3. Postoperative roentgenogram of control dog 19 showing shift of mediastinum

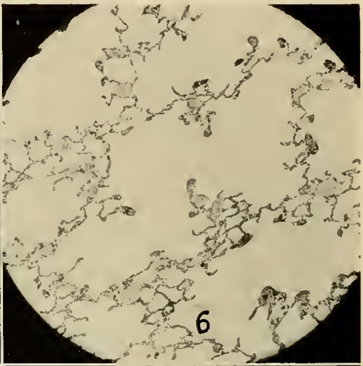
Fig. 4. Roentgenogram of experimental dog 24 with mediastinum close to midline

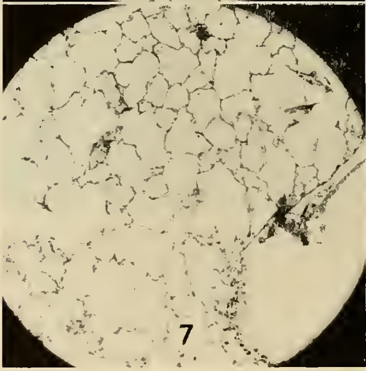
Postoperatively the resected lung tissue was immediately evacuated of air and the volume measured by the water displacement method. Air was then injected into the pulmonary tree until a pressure of 10 mm. of mercury[3] was reached and this volume recorded. Formalin was instilled into the bronchi. The entire specimen was immersed in formalin and kept distended constantly at 10 mm. of mercury pressure and allowed to fix in this state for a period of 24 hours.

Later the dogs were sacrificed in such a manner that the animal's final breath was at the end of inspiration. This allowed for measurement of the thoracic cavity under constant conditions.

After the dog was anesthetized it was placed exactly in dorsal position. While being bled from the femoral artery a 10 per cent formalin solution was introduced under pressure into a vein in the extremity. At the last inspiratory effort a clamp was placed across the previously isolated trachea. The body was allowed to harden for several hours. The remaining lung tissue was carefully dissected out of the thorax; the other thoracic organs being left in situ. The volume of the thoracic cavity was







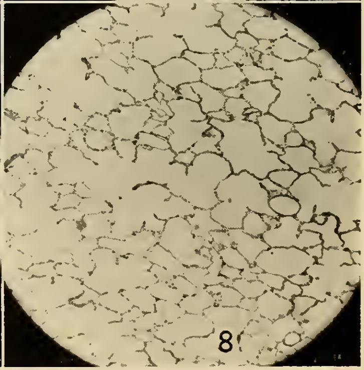


FIG. 5. Section of postoperative specimen of control dog 17, showing normal pulmonary structure

FIG. 6. Autopsy section of control dog 17 demonstrating disruption of alveolar walls with fusion of the ends.

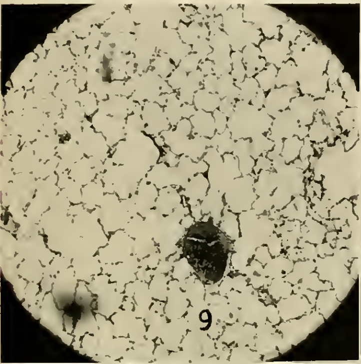
FIG. 7. Resected specimen of experimental dog 25 with normal parenchyma

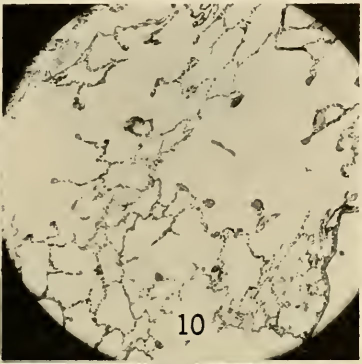
FIG. 8. Autopsy specimen of experimental dog 25 showing normal lung parenchyma

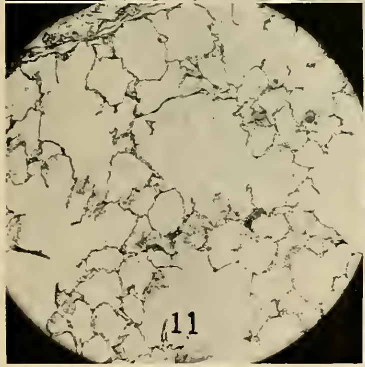
then measured and recorded. The resected pulmonary tissue was treated in the same manner as described above, except that the air was not compressed out but its volume was measured in the distended state.

Sections of lung tissue were carefully cut for microscopic study so that there would be no distortion or compression of the alveoli in manipulation.

The lobectomized dogs were treated in the following manner. After anesthetization a pneumonectomy was performed on the intact side. Following this, formalin was infused into the dogs' veins and treated as previously stated. The intact lung was aspirated, the volume measured, and the volume of air necessary for normal distention recorded. The specimen was then fixed in the distended state for 24 hours.







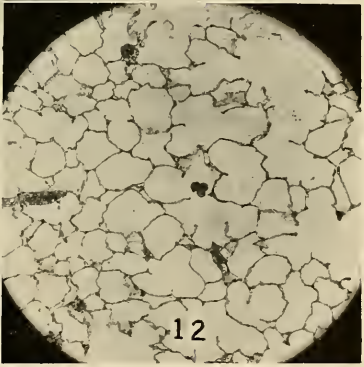


Fig. 9. Section of surgical specimen of control dog 19 showing normal structure

Fig. 10. Autopsy section of control dog 19 illustrating clubbing of broken ends of alveoli

Fig. 11. Surgical specimen of experimental dog 24 showing minimal interalveolar fragmentation

Fig. 12. Autopsy specimen of experimental dog 24 revealing minimal interalveolar fragmentation

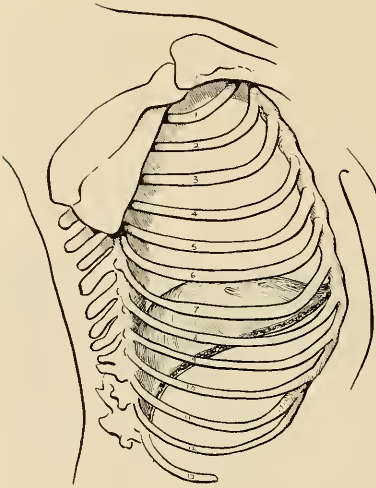


Fig. 13

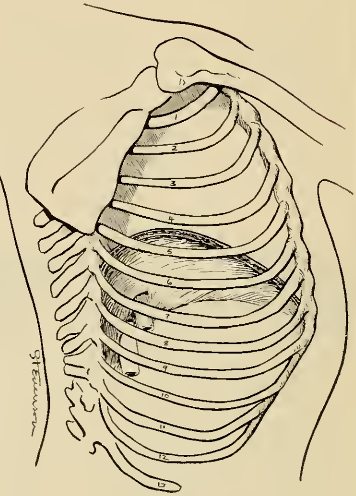


Fig. 14

Fig. 13. Normal attachment of dog's diaphragm

Fig. 14. Depicting freeing of diaphragm along anterior, lateral, and most of posterior surfaces and elevation to a higher level.

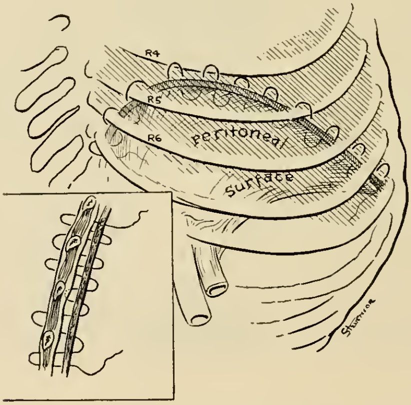


Fig. 15. Showing method of suture

The volume of the remaining lobe or lobes was measured in the distended state and fixed for 24 hours. Specimens were taken for microscopic study of the lung tissue and diaphragms.

Preoperative and postoperative chest roentgenograms were routinely made and a fluoroscopic study relative to the position and motion of the diaphragm was recorded on all animals.

## OPERATIVE PROCEDURE

The sixth or seventh interspace was usually entered. In the experimental dogs the diaphragm was dissected completely free in the anterior, lateral and most of the posterior portions. The detached diaphragm was then reattached to a higher level (Figs. 13 and 14). The diaphragm was sutured to the thoracic wall by means of a nonabsorbable material. The suture was run through the edge of the diaphragm, from the peritoneal surface to the pleural surface, then to the pleural surface of the thoracic wall, and finally through the intercostal muscle bundle and back again (Fig. 15). The diaphragm was relocated to a level as high as the fourth interspace without too much tension on the suture line.

## RESULTS

In the two control pneumonectomized dogs, No. 17 and No. 19, microscopic examination revealed the following: The postoperative specimen of dog No. 17 (Fig. 5) showed normal pulmonary structure with small air sacs and large ducti. The autopsy section (Fig. 6) revealed true emphysema with disruption of the alveolar walls and a significant fusion and clubbing of their broken ends. In dog No. 19 (Fig. 9) there was normal lung parenchyma in the resected specimen and emphysema with clubbing in the autopsy sections (Fig. 10).

Study of the microscopic section in dog No. 25 of the experimental pneumonectomized dogs revealed normal parenchyma in the resected specimen (Fig. 7) and in the autopsy specimen (Fig. 8). In dog No. 24 the caliber of the alveoli appeared to be the same in both sections and showed minimal clubbing. The autopsy specimen (Fig. 12) revealed no more clubbing than existed in the surgical section (Fig. 11).

At autopsy the lobectomized control dogs showed in general a dilatation of the remaining lung tissue in comparison with the postoperative specimen. In dog 42 there was a small amount of clubbing present in both the postoperative and autopsy specimens. There was a greater degree of dilatation of the alveoli in the latter specimen than in the former. Experimental dog No. 27 revealed normal lung parenchyma in the postoperative specimen and in the autopsy sections. There was a great difference between the two sections in dog 26. The postoperative specimen showed normal appearing parenchyma. In the autopsy section dilatation and clubbing were seen. Experimental dog No. 28 revealed moderate emphysema and peripheral dilatation, with deeper normal structure in the autopsy sections.

Dog No. 59 showed alveoli of normal caliber with some pre-existing clubbing in the postsurgical specimen. The autopsy section showed clubbing at the periphery with distention of the alveoli. In experimental dog No. 29 the alveoli and ducti were

Table I

| | | | VOLUME STUDIES (CC'S) | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | RESIDUAL TISSUE | | | | | | | | | | | | |
| | | | | ESTIMATED • OR MEASURED | | | | | | | | | | | | |
| | | | | CONTRALATERAL | | IPSILATERAL | | | | | | POSITION OF DIAPHRAGM | | | | |
| DOG NO. | | OPERATIVE PROCEDURE | RESECTED PULMONARY TISSUE | SURGICAL OR AUTOPSY | THEORETICAL | SURGICAL OR AUTOPSY | THEORETICAL | PERCENTAGE OF VOLUME INCREASE OF RESIDUAL TISSUE | PLEURAL CAVITY | PLEURAL CAVITY ESTIMATED | POSITION OF MEDIASTINUM | POST-OP | MOVEMENT | PHRENIC CRUSH | POST-OP PERIOD | WEIGHT LBS |
| 17 | C | PNEUMONECTOMY LEFT | 1050 | 1990 | 1280 | | | 55% | 2300 | 2330 | TOWARD OPERATIVE SIDE (FIG 1) | 8-9TH INTERSPACE | I INTERSPACE | – | 6 MONTHS | 30 |
| 25 | S | PNEUMONECTOMY LEFT + RD | 1220 | 1650 | 1490 | | | 10 | 1900 | 2710 | MIDLINE (FIG 2) | 4TH IS | NONE | + | 6 MONTHS | 32 |
| 42 | C | UPPER LOBECTOMY LEFT | 320 | 950 | | 720 | 770 | 33 | 1720 | 1730 | MIDLINE | NORMAL | I-IS | – | 6 MONTHS | 26 |
| 27 | S | LUL + RD | 330 | 980 | | 580 | 800 | 12 | 1500 | 1780 | MIDLINE | 5TH IS | I-IS | – | 6 MONTHS | 28 |
| 26 | C | LOWER LOBECTOMY LEFT | 740 | 1120 | | 400 | 916 | 24 | 2000 | 2040 | MIDLINE | NONE | I-IS | – | 5 MONTHS | 26 |
| 28 | S | LLL + RD | 460 | 840 | | 240 | 690 | 18 | 1100 | 1530 | MIDLINE | 5TH IS | I-IS | + | 6 MONTHS | 22 |
| 19 | C | PNEUMONECTOMY RIGHT | 1250 | 1700 | 1022 | | | 66 | 2250 | 2270 | TOWARD OPERATIVE SIDE (FIG 3) | NORMAL | I-IS | – | 6 MONTHS | 32 |
| 24 | S | PNEUMONECTOMY RIGHT + RD | 1340 | 1210 | 1096 | | | 10 | 1440 | 2440 | MIDLINE (FIG 4) | 4TH IS | DIFFICULT TO DETECT | – | 6 MONTHS | 33 |
| 59 | C | UPPER LOBECTOMY RIGHT | 310 | 870 | | 850 | 1060 | 91 | 1990 | 1930 | MIDLINE | NORMAL | I-IS | – | 6 MONTHS | 25 |
| 29 | S | RUL + RD | 330 | 780 | | 630 | 950 | 72 | 1320 | 1730 | MIDLINE | 5TH IS | I-IS | – | 6 MONTHS | 22 |
| 4 | C | LOWER LOBECTOMY RIGHT | 640 | 780 | | 420 | 950 | 112 | 1700 | 1730 | MIDLINE | NORMAL | I-IS | – | 6 MONTHS | 22 |
| 5 | S | RLL + RD | 670 | 900 | | 450 | 1100 | 18 | 1510 | 2000 | MIDLINE | 5TH IS | I-IS | + | 6 MONTHS | 24 |

C Control
S Subject
* 55% for right lung, 45% for left
RD Relocation of diaphragm
UL Resection of apical and cardiac lobes
LL Resection of diaphramatic and intermediate lobes
Standard diviation—minimum of 20 cc.

larger than in the autopsy specimen, in which the alveoli appeared to be normal. Sections of control dog No. 4 showed normal parenchyma in comparison to autopsy specimens, which showed large ducti and fragmentation of the alveoli, with moderate clubbing. Comparison of the surgical and autopsy sections revealed an equal status of the alveoli and ducti, with some fragmentation.

## VOLUME STUDIES

The percentage of volume increase of the residual pulmonary tissue of control dogs No. 17 and 19 amounted to 55 per cent and 66 per cent respectively (Table I). This totaled 10 per cent and 10.4 per cent in experimental dogs No. 25 and 24. The volume increase in the upper lobectomies of the controls (No. 42 and 59) amounted to 33 per cent and 9.1 per cent. The lower lobectomies showed 24 per cent and 11.2 per cent. The corresponding experimental dogs showed volume increases of 12.0 per cent, 7.2 per cent, 1.8 per cent and 1.8 per cent.

## SUMMARY

The formation of a true emphysema after pulmonary resection has occurred to some degree in the six dogs used as controls, especially where pneumonectomies have been performed. This was evidenced by the presence of dilated alveoli with fragmentation of their walls and subsequent fusion of the broken ends. To a more limited extent, this pathologic condition has developed after lobectomies.

The procedure of relocating the diaphragm following resection has noticeably resisted the sequelae of overdistention and emphysema. This result was especially evidenced after pneumonectomies, where the gross changes are more likely to occur.

Volume changes, though not performed under absolutely scientific methods, have resulted in a substantial reduction of pulmonary dead space by relocation of the diaphragm.

## CONCLUSIONS

1. A new method of obliterating pulmonary dead space is presented.
2. Relocation of the diaphragm after pulmonary excision has prevented or diminished overdistention and/or emphysema.

### *Note*

Since the inception of this work relocation of the diaphragm has been performed on 2 patients and will be reported elsewhere.

### *Acknowledgement*

The authors wish to thank Dr. C. Gardner Warner, pathologist at Mercy Hospital, Baltimore, for interpreting the microscopic sections.

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# CLINICO-PATHOLOGIC CONFERENCE

### From the Case Histories, University Hospital, Baltimore

### *Clinical History*

In May, 1949 a 54 year old white male, had what was called "virus infection of the lungs." At that time, a chest roentgenograph was reported as negative. Illness that was consistently diagnosed as virus respiration infection recurred several times. In February and March, 1951, the patient was bedridden for 7 days, with a fever, cough, malaise, and generalized aches. On October 27, 1951, noting that he was warm, and finding his temperature to be 102 F., his wife called the family doctor. Abnormal sounds were found in the right hemithorax. The patient was kept at home under observation and treated with aureomycin. His temperature ranged between 100 F. and 102 F. After 5 days, he returned to work, but his temperature rose to 104 F. He again went to bed and was treated with chloromycetin. Erythrocytes and albumin were found in his urine. A roentgenograph revealed a shadow in the right lower lobe. Since that time (November 13), he had not returned to work. The urine cleared, he became asymptomatic and the fever gradually subsided. Even though he currently felt well on December 17, 1951 he entered the University Hospital.

When seen by the examiner, this patient was alert, cooperative, and apparently comfortable. He was slightly obese and florid. His body temperature was 99.2 F.; pulse rate, 90 per minute; and respiratory rate, 24 per minute. Nothing significant was found by examination of the upper respiratory and cardiovascular systems. The note elicited by percussion of the right lung base was impaired. Here, the breath sounds, though pure, were distant. The excursion of this base was diminished. Irrelevant trivia were disclosed by examination of the abdomen, genitalia and extremities.

The laboratory data were unfruitful. The leucocytes, though not increased in number, were 14 per cent eosinophiles. The gastric washings and sputum were free of tubercle bacilli. Pyelograms and roentgenographs of the chest were taken.

By bronchoscope, fixation and moderate spastic narrowing of the bronchus to the right lower lobe were noted. The mucosa was reddened. A globoid lesion was found and removed by lobectomy. Thirteen days later the patient left the hospital feeling well and apparently cured.

### *Clinical Discussion*

*Dr. R. A. Cowley:* For nearly 3 years, this man periodically has been assailed by fever, cough and malaise. At first the sickness was called virus pneumonitis, but chronicity moves against this impression. One should always be alert to the dangers of mistaking a neoplasm or infectious granuloma for virus pneumonitis. A precautionary rule of doubting the accuracy of a diagnosis of virus pneumonia is urged when the disease persists for more than 3 weeks. Bronchiogenic carcinoma, tuberculosis or fungus infection should be considered in such cases.

Carcinoma is unlikely in this patient. He had the disease for $2\frac{1}{2}$ years, yet he was well nourished, not anemic, and free of metastases as far as one could tell. There were no tumors in the skin or in the cervical lymph nodes, and there were no evidence of visceral or encephalic metastases.

Tuberculosis, if active for 2 years, surely would show signs of its progression in serial roentgenographs and probably would permit more than the minimal symptoms reported here. This is also true of mycotic infections. I should suspect that cultures of sputum or gastric washings in this case would be negative. The existence of this lesion in the right base rather than apex is not conducive to the diagnosis of phthisis.

The eosinophilia reported here demands at least a mention of Loeffler's syndrome. but again the remittent course of this malady and the bronchoscopic report preclude this possibility. Also, it is suspected that the radiographic studies of this lesion will show it to be more or less stationary. This is not the case in Loeffler's disease.

A casual notice is given aspirated foreign body and abscess. These afflictions usually cause a more dramatic series of symptoms. Hectic fever and leukocytosis would be demonstrated; asthma may be associated. If bronchiectasis were present, I assume that it occurs as a complication of a localized lesion. This assumption will probably be supported by roentgenography.

The hematuria reported in this case is difficult to explain. Metastatic carcinoma of the kidney might be incriminated, but, to my mind, this possibility should be dropped for the reasons given for the exclusion of bronchiogenic carcinoma. If one were to consider the pulmonary lesion as a metastic one, the kidney could be named as the primary site. I feel that an intravenous pyelogram would be normal. Primary sites in systems other than the lung are surely not indicated by the signs and symptoms ascribed to this patient. Relevant to the theory that he had a localized pulmonary lesion, renal emboli arising from pulmonary venous thrombi might be culpable. Venous thrombosis in the environs of the pulmonary lesion seen in this man's right lower lung lobe is merely hypothetical.

Periodicity, chronicity without debility, absence of evident dissemination, and presumed localization suggests to me the diagnosis, bronchial obstruction caused by bronchial adenoma.

*Dr. Charles N. Davidson:* This film, taken two days after admission (see illustrations) like previous ones shows an area of diminished radiolucency which converges toward the hilus. This is taken to be an area of atelectasis. The lymph nodes in the hilus are of normal size. There were no significant changes seen in the pyelogram.

*Senior Student:* Do you think this patient mightn't have pulmonary adenomatosis?

*Dr. Cowley:* Pulmonary adenomatosis is more characteristically bilateral. Even if it is unilateral many lesions are seen and not one as in this case.

*Senior Student:* Fungus infection and carcinoma are not completely ruled out.

*Dr. Cowley:* That is true, but for the reasons cited I favor the diagnosis, bronchial adenoma.

### *Pathologic Discussion*

*Dr. Hugh R. Spencer:* The gross specimen in this case consists of a portion of the right lower lobe bronchus, with lung tissue attached. Projecting into the lumen of the bronchus there is a rounded polypoid mass measuring 1.3 cm. in diameter. The mass is external to the cartilage, it is covered by intact mucosa. On section, it is composed of minute cystic areas filled with mucoid material. The lesion appears well circumscribed.

Sections are made, including the wall of the bronchus and the tumor. The tumor is composed of closely approximated glandular structures, each of which is filled with mucin. The lining cells are tall cylindric in shape, and the nuclei are situated at the bases of the cells. The lesion is well circumscribed. It is not invasive. It lies between the cartilage and the mucosa, and it is covered by an intact mucous membrane.

Adenoma of the bronchus is a "slowly growing, at first circumscribed, but later sometimes invasive or sluggishly metastasizing polypoid or sessile epithelial neoplasm involving the subepithelial tissues usually of proximal bronchi" (Liebow). Several histologic types are described. These are 1. Carcinoma; 2. "Mixed tumor type"—basal cell or cylindroid type; and 3. Adenoma, mucous glands. While the exact origin of these tumors is not entirely clear, the opinion held by most is that they originate from mucous glands and mucous gland ducts. The classification as to type is not an exact one. Some of the tumors present varied histologic aspects.

Bronchial adenomas are slowly growing, polypoid or sessile tumors found usually in the large bronchi. They make up from 6 to 10 per cent of all the primary lung tumors. The lesions are generally regarded as benign, although in a small percentage of cases invasion of the hilar and mediastinal lymph nodes is noted. Metastases to the liver, vertebrae and kidney have been described. Some classify bronchial adenomas as Grade I carcinomas. The carcinoid type is the most common and the cylindroid type is more apt to invade. The carcinoid type is generally radioresistant. The cylindromatous type may show regression following roentgen ray therapy.

Of the patients who exhibit this type of lesion, 85 per cent are under fifty years old. The average age at the time of treatment is from thirty-five to forty years. The usual symptoms are wheeze, cough, hemoptysis. Recurrent pneumonitis and bronchiectasis may be complicating factors.

### *Anatomic Diagnosis*

Adenoma, right lower lobe bronchus, mucous gland type.

## BOOK REVIEWS

*The Practice of Balanced Anesthesia.* By SYLVAN M. SHANE: Attending Anesthesiologist, Lutheran Hospital of Maryland. Cloth. Price $5.75. Pp. 221, Baltimore, Md., Lowry and Volz, Publishers, 1953.

This book contains a wealth of practical information and can be used as an excellent guide to anesthesiology. It is divided into four main divisions. The first details the use of a synergistic balanced anesthesia combination. Its formula and technique are devised by the author and labeled "The Balanced Mixture".

The proportions of the "Balanced Mixture" consist of Cyclo-propane-300 cc. per minutem nitrous oxide-1000 cc. per minute, and oxygen-2000 cc. per minute. These figures are multiplied by two if administered to children under the age of ten. To either child or adult these gases are administered via a semi-closed, carbon dioxide absorption system, wherein a portion of each exhalation is blown out into the operating room. This obviates the portent of carbon dioxide and heat retention, two of the undesirable complications of closed system anesthesias. The author, in fact, condemns the closed system as an antiquated method of general anesthesia.

This mixture of gases, however, will not maintain anesthesia by itself, nor is it the complete "Balanced Mixture". The patient must first be rendered unconscious by some short acting barbiturate, such as Evipal® or Pentothal®. Following the induction with Evipal, for example, the gaseous mixture of nitrous oxide, cyclopropane and oxygen will maintain plane one anesthesia indefinitely without the necessity for any additional barbiturate, regardless of the time consumed in surgery. The "Balanced Mixture", then, is defined as the combination of an initial and singular dose of a barbiturate plus a specific gaseous combination of nitrous oxide, cyclopropane and oxygen, administered in a semi-closed system. Since anesthesia is so light, some form of curare must be utilized to provide relaxation.

The book reports over 15,000 human administrations of the "Balanced Mixture" for every type of surgery, without a fatality resulting from anesthesia. This reviewer can attest to the superiority of the "Balanced Mixture" anesthesia since his results have been identical with that claimed by the author of the book. The chief advantages claimed for the "Balanced Mixture" over any other type of general anesthesia appear to be the speed with which patients react post-operatively—often within a matter of minutes, even after surgery requiring many hours. Another advantage is the relative absence of post-operative hypotension seen especially after prolonged closed system anesthesia, and-also the infrequency of palpable cardiac arrythmias.

The first section also covers the administration of the "Balanced Mixture" for intubation, eye, ear, nose and throat, neuro, thoracic, obstetric, dental and pediatric surgery. There is also a chapter on its use in small animal surgery.

The second section on "Balanced Regional Anesthesia" covers the single and continuous spinal methods in reference to surgery and obstetrics, and explains methods of combining both regional and general anesthesia in the same patient.

The third section presents chapters on the asthmatic patient under anesthesia, blood transfusion reactions, fluid therapy during surgery, and the treatment of cardiac arrest. Each of these problems is discussed in an orderly fashion and the methods recommended to cope with them are treated fully.

The final section of the book is devoted to an interesting and most enlightening discussion on the discovery of anesthesia, the pioneers in this field, their historical background, and their contribution towards the evolution of balanced anesthesia in general.

*I. William Towlen, M.D.*

**Clinical Heart Disease.** *Samuel A. Levine, M.D., F.A.C.P., Clinical Proflsor of Medicine, Harvard Medical School; Physician, Peter Bent Brigham Hospital, Boston; Consulant Cardiologist, Newton-Wellesley Hospital, Physician, New England Baptist Hospital. W. B. Saunders Company, Philadelphia & London, 1951. Fourth Edition. 556 pages. Illustrated. Price 7.75.*

The fourth edition of Dr. Levine's practical and instructive text dealing with the heart and it's diseases contains in its revision, many of the advances made in the fields of diagnosis and treatment of heart disease which have appeared since the highly regarded 1945 edition. In the present edition, as in previous ones, the author writes with authority on his subject and occasionally, with genuine affection when discussing his favorite aspects of heart disease. Nevertheless, in doing so, he does not neglect other perhaps less interesting but none the less essential features of the subject.

The book is divided into 21 well written chapters dealing with most aspects of clinical heart disease of interest to the practitioner of medicine. The ever popular section devoted to electrocardiography has been revised and expanded to include a discussion of unipolar leads and other diagnostic aids developed in recent years. The illustrations present in the book are confined to the section on electrocardiography, and consist of exemplatory tracings.

As in previous editions, the author devotes a short but important chapter to the recognition and differentiation of functional heart disease. Emphasis is placed on the responsibility of the physician in the prevention of many iatrogenic heart conditions. The chapters concerned with acquired heart disease have been re-written to include a frank appraisal of the much heralded surgical approach to chronic valvular disease. As in the past, the value of the text is enhanced by a modest but concise index.

The acquisition of this book is recommended to the active practitioner of medicine, not only for the timely and complete contents but particularly for the way in which the subject is presented, making it quite readable.

J. B. W.

**The Practice of Endocrinology.** *Edited by Raymond Greene, M.A., D.M., M.R.C.P. (London), Physician, the Metropolitan Hospital, Royal Northern Hospital, Harrow Hospital and the Thyroid Clinic, New End Hospital, Hampstead, England; formerly Hunterian Professor Royal College of Surgeons of England. J. B. Lippincott Company, Philadelphia, London and Montreal. 1951. Second Edition. 389 pages; illustrated. Price 12.50.*

The Practice of Endocrinology was written by the senior author and his collaborators with the purpose of presenting the essential features of endocrinology in a form suitable to the average busy practitioner of medicine. For this reason, many unproven hypotheses and considerable academic detail were purposefully omitted and emphasis placed on the diagnosis and treatment of endocrine disorders.

The book was first published in England during 1950 but was not well received because of excessive detail. In 1951, Dr. Greene revised the earlier work by deleting much of the questioned material and published the remainder in the present form as a second edition.

In general, the book consists of 9 rather large chapters each of which considers an important endocrine organ or disorder in detail. The chapters begin with a review of the anatomy and physiology of the organ under discussion and conclude with disorders of the gland, their recognition, differential diagnosis and therapy. The sections devoted to disorders of the thyroid and to the adrenal gland are especially well written while the chapter concerned with diabetes is less well constructed. Numerous illustrations, charts and graphs enhance the understanding of the printed material. In addition, a well selected bibliography is appended to each chapter and a subject index appears at the end of the book.

In the opinion of the reviewer, the authors have accomplished their purpose in presenting the essentials of endocrinology in an attractive and understandable manner. In truth, the text is well titled for it is written for the physician interested in the PRACTICE of endocrinology.

J. B. W.

# MEDICAL SCHOOL SECTION

## NEW ADMINISTRATIVE OFFICE CREATED BY BOARD OF REGENTS

*Position of Director of Medical Education and Research Created*

By action of the Board of Regents of the University of Maryland, the office of Director of Medical Education and Research was established for the purpose of coordinating and promoting the efficiency of the School of Medicine, the School of Nursing and the University Hospital in all phases of their respective work.

In a directive issued to members of the Faculty the Administrative Board stated that the Deans of the Schools of Medicine and Nursing and the Director of the Hospital were to maintain their prerogatives in directing the respective agencies under their jurisdiction but that the ultimate authority in directing these three associated groups would now rest under the direction and advice of this newly created administrative official. The Director of Medical Education and Research in turn is responsible to the President of the University.

Continuing, the Board of Regents directed that the Director of Medical Education and Research advise and plan future developments and policies in the educational research activities of the medical agencies. Likewise, he has been given the responsibility for the submission of the respective budgets for these agencies. All recommendations for major personnel appointments shall be submitted through him to the President of the University.

This new office carries the responsibility for further extension and development of the physical plant, for development of a Planning Committee for these agencies in Baltimore City. His duties also include the participation in meetings of students and faculties of the respective agencies and these faculties are instructed to turn to him for advice such as is appropriate for the academic development of the schools.

The Director will also be the official representative of the University to the American Medical Association, the Association of American Medical Colleges and the Maryland State and Baltimore County Medical Societies.

## DR. HACHTEL RETIRES

Dr. Frank Wilson Hachtel, Maryland Medical College, class of 1904 and Professor of Bacteriology at the School of Medicine since 1924 retired on July 1, 1954. Dr. Hachtel's period of service, one of the longest among members of the medical faculty covers a span of nearly 50 years.

Following his graduation from the public schools of Baltimore and the Maryland Medical College, Dr. Hachtel served a year of internship at the Franklin Square Hospital and an additional year at the United States Marine Hospital. He then became a member of the professional staff of the Baltimore City Health Department serving as an assistant City bacteriologist. At this time he was associated with the late Dr. William Royall Stokes, who was the founder of the Bacteriologic Division

Dr. Hachtel—familiar pose

of the Baltimore City Health Department. In this capacity he served until 1908. He was associated for a brief period with the United States Public Health Service and helped organize the Bacteriologic Division of the Health Department in the City of Detroit. In 1913 he returned to the Baltimore City Health Department. Dr. Hachtel served abroad with the United States Army in 1917. In 1920 he left his post at the Baltimore City Health Department. He returned to the School of Medicine as Associate Professor of Bacteriology, becoming Professor of Bacteriology in 1924.

Dr. Hachtel's interests in bacteriology have been diverse and included many fundamental researches now considered an integral part of bacteriologic science. He was one of the pioneers in the use of radiation as regards its effect on micro-organisms, being the author of a paper entitled "The Effects of Radium Irradiation on Electrophoretic Velocity, Viability and pH of Escherichia coli Suspensions."

Dr. Hachtel has also investigated extensively the mechanisms of epidemic spread of diphtheria and epidemic meningitis. In addition he contributed to fundamental researches concerning the metabolic activity of bacteria with respect to changing aspects of environment.

During the academic year 1953–54 members of the faculty honored Dr. Hachtel with a testimonial luncheon at the University Hospital and presented him with a handsome radio-phonograph.

Still in excellent health, Dr. Hachtel has earned a long sabbatical leave. He has informed us that he intends to proceed with his reading and continued study as has been his custom.

## DR. WILLIAM S. STONE APPOINTED DIRECTOR OF MEDICAL EDUCATION AND RESEARCH

Dr. William Spencer Stone has been recently named by the Board of Regents of the University of Maryland as Director of Medical Education and Research. Dr. Stone has come to the University of Maryland from the Walter Reed Army Medical Center where, since 1950 he has served as Commandant of the Army Medical Service Graduate School with the rank of Colonel in the Medical Corps, United States Army.

Dr. Stone was born in Ogden, Utah on March 20, 1902, and is an alumnus of the University of Louisville School of Medicine, class of 1929. After his graduation he received his basic clinical training at the William Beaumont General Hospital at El Paso, Texas, entering the United States Army Medical Corps in 1929 with the rank of 1st Lieutenant.

From 1934 to 1938 he was Chief of the Protozoology and Virus Disease Section of the Army Medical School following which he became a member of the Army Medical Research Board at Ancon, Canal Zone serving from 1938 to 1939. During the next year he served as Bacteriologist at the Board of Health in Panama becoming Chief of the Sanitation and Laboratories Division in the Office of the Surgeon General from 1941 to 1943. During this time he contributed a number of original scientific papers relating to the encystment of endamoeba histolytica and the staining of protozoa.

Dr. William S. Stone

In 1943 he became Consultant and Chief of Preventive Medicine in the Mediterranean Theater. He then became Chief of the Preventive Medicine Division, Air Surgeon's Office. From 1946 until 1950 he was Chairman of the Medical Research and Development Board, Office of the Surgeon General and was Army Representative to the National Research Council also during this period. From 1950 until his appointment to the University of Maryland he was Commandant of the Army Medical Service Graduate School of the Walter Reed Army Medical Center.

In addition to Dr. Stone's scientific investigations with respect to tropical diseases and protozoa he has contributed a number of scientific articles relating to the treatment of thermal burns and basic scientific studies relating to radiation on microorganisms.

He has also served as Director of the Army medical in-service research programs in the fields of communicable diseases, surgery of trauma, metabolism and stress. For this and other scientific contributions he has received the Sternberg Medal, the Legion of Merit with Oak Leaf Cluster, the Typhus Commission Medal, the Order of the British Empire and an honorary Doctor of Philosophy degree from the University of Louisville in 1948. He is a diplomate of the American Board of Pathology (Clinical Pathology) and the American Board of Preventive Medicine and Public Health. He is a member of Sigma Xi and Alpha Omega Alpha. He is a fellow of the American Medical Association, Fellow of the New York Academy of Medicine, New York Academy of Sciences, District of Columbia Academy of Medicine, the American Society of Tropical Medicine and the Aero Medical Association.

## DR. PINCOFFS APPOINTED PROFESSOR OF PREVENTIVE MEDICINE

### *Resigns as Professor of Medicine to Form New Department*

Dr. Maurice C. Pincoffs, Professor of Medicine at the School of Medicine, resigned as of July 1, 1954 to accept the newly created Professorship of Preventive Medicine and to begin organization of this department. This important appointment and the challenge it offers has been accepted by a man who has devoted a long and brilliant career to the service of the School of Medicine.

Dr. Pincoffs received his Bachelor of Science degree from the University of Chicago in 1909 and his degree of Doctor of Medicine from the Johns Hopkins University in 1912. From 1915 until 1917 he was Instructor in Medicine at the Johns Hopkins University School of Medicine, interrupting his early training to enter World War I as a Lieutenant in the Medical Reserve Corps, being first assigned to active duty with the British Army. Eventually Dr. Pincoffs became battalion medical officer with the 9th Infantry and later commanding officer of a Medical Company.

Upon his return to Baltimore he was appointed Professor of Medicine and Chairman of the Department of Medicine at the University of Maryland School of Medicine, assuming this post in 1922. Since that time Dr. Pincoffs has had a distinguished career in medical science, as an editor, an organizer of the very fine Department of Medicine and as an advisor to many National Organizations and Governmental Agencies. He has long been a member of the Maryland State Board of Health and Chairman of the Committee on Medical Care of the Maryland State Planning Committee. He has advised the Secretary of War and was formerly a member of the Armed Forces Medical Advisory Council. In 1951 he was elected President of the American College of Physicians and currently serves as editor of the official College publication "The Annals of Internal Medicine".

Dr. Maurice C. Pincoffs

In World War II Dr. Pincoffs organized the 42nd General Hospital as a University of Maryland Unit and served with distinction during the entire World War serving as Chief Medical Consultant in the office of the Surgeon General, Far Eastern Command. After the war he continued as a Reserve Officer until he retired in 1949. In 1953 he was nominated a member of the Advisory Council of the National Institute of Arthritis and Metabolic Diseases.

Dr. Pincoffs' distinguished career and wide international experience, his familiarity with the cause and prevention of disease, his activity with governmental and national associations, plus his long career as a consultant and advisor becomes now most valuable as he proceeds to organize the new Department of Preventive Medicine.

## DR. THEODORE E. WOODWARD NAMED PROFESSOR OF MEDICINE

*Assumes Departmental Chairmanship as Successor to Dr. Pincoffs*

Dr. Theodore E. Woodward, an alumnus of the School of Medicine, class of 1938, was recently named Professor of Medicine and Chairman of the Department. He succeeds Dr. Maurice C. Pincoffs, who tendered his resignation to become the first Professor of Preventive Medicine, effective July 1, 1954.

Dr. Woodward, who is well known for his contributions to the knowledge of rickettsial, virus and bacterial infectious diseases and for his contributions to the therapy of typhoid fever and Rocky Mountain spotted fever was born on March 22, 1914 in Westminster, Maryland. A graduate of Franklin and Marshall College in 1934, he completed his medical studies at the University of Maryland and following his graduation served as a rotating intern at the University Hospital from 1938–40. During the year 1940 he was an assistant resident at the Henry Ford Hospital in Detroit where his chief interest centered in gastrointestinal diseases.

Dr. Theodore E. Woodward

As a member of the Officers' Reserve Corps he was called to active duty in February, 1941 and was immediately assigned to Fort Meade. From Fort Meade he entered the School of Tropical Medicine in Washington and then was assigned to Bermuda for field work in Dengue fever. While he was assigned to service in Jamaica, British West Indies, he became interested in virus and rickettsial diseases. He was assigned thereafter to the Army Medical School, Virus and Rickettsial Department, participating in the North African Campaign and later as a member of the United States Army Typhus Commission. During this tenure he studied Malayan scrub typhus during the year 1948.

After his discharge from the Army of the United States with the rank of Lieutenant

Colonel he returned to the School of Medicine as Director of the Section of Infectious Diseases, University of Maryland School of Medicine. He was then successively Assistant Professor of Medicine, Associate Professor of Medicine and recently Professor and Chairman of the Department. Dr. Woodward also holds appointments as a Consultant in Internal Medicine to the State Health Department, Lecturer in Preventive Medicine at the Johns Hopkins School of Medicine and Consultant to the Veterans Administration. He is a member of the Sub-Committee on Infectious Diseases and Chemotherapy, National research Council. He is Consultant, Walter Reed Medical Center and Regional Director, Sectional Research Program of Microbiology, United States Public Health Service. He is Associate Member of the Commission of Hemorrhagic Fever, Army Medical Service Graduate School, Member of the Commission of Immunization, Army Medical Service Graduate School, Member of the Plague Commission, United States Army, a Consultant of the Army Chemical Center in Maryland, Member of the Society of Medical Consultants to Armed Forces, Member of the Medical Services to Maryland Civil Defense, Consultant to the Surgeon General's Office, Washington, D. C. and a member of numerous learned societies.

In 1950, Western Maryland College honored him with the Doctor of Science degree.

Dr. Woodward is a member of the Sigma Pi collegiate fraternity, Nu Sigma Nu and an honorary member of Alpha Omega Alpha Society. His bibliography includes over 60 titles, the majority concerning infectious diseases, particularly virus and rickettsial diseases.

Dr. Woodward is a forceful speaker and an intensely enthusiastic investigator. His interest in medical education has been recognized by his appointment as Chairman of the Curriculum Committee of the Medical School, a Committee which has been charged with the complete revision of the basic teaching program and with the coordination of numerous departments in accordance with latest and most acceptable techniques.

His appointment brings to the Department of Medicine a competent teacher, enthusiastic clinician and a physician most superbly trained and constituted to lead the department's research program and to provide a signified and scholarly academic program.

## UNIVERSITY OF MARYLAND BIOLOGICAL SOCIETY

The Bulletin announces the resumption of a departmental feature, the Proceedings of the University of Maryland Biological Society. This section of the Bulletin is separately captioned and contains the Proceedings of the University of Maryland Biological Society and abstracts of pertinent research. The first abstract is herewith presented.

# POST GRADUATE COMMITTEE SECTION

## POST GRADUATE COMMITTEE, SCHOOL OF MEDICINE

HOWARD M. BUBERT, M.D., *Chairman and Director*
Elizabeth Carroll, *Executive Secretary*
Post Graduate Office: Room 600
29 South Greene Street
Baltimore 1, Maryland

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## TELEVISION IN RETROSPECT

### *"Live and Help Live" Completes Third Year*

On May 24, 1954 "Live and Help Live" the University of Maryland's public service scientific program, presented over WBAL-TV Monday evenings, came to a close.

Again, the program has been under the capable directorship of Anne Holland and received excellent support from the staff and management of WBAL-TV, National Broadcasting affiliate (channel 11) in Baltimore.

Acting upon requests and with the experience of previous years, unit programs were first introduced. The subject of heart disease was covered by a panel of physicians beginning on October 26 with anatomic and physiologic concepts of the heart presented. On November 2—the subject was continued with rheumatic heart disease. The program on November 9, 1953 concluded with a telecast on the failing heart, surgical treatment, and the prevention and control of heart disease. These integrated units, useful in presenting in more detail long and complex subjects, were very successful and will constitute a nucleus for further development in the 1954–55 planning.

Members of the staff of the School of Medicine, the Schools of Nursing, Dentistry, Pharmacy and the staff of the University Hospital are to be complimented on a careful preparation of material and a thoughtful presentation. This planning resulted in a program maintaining steady public interest despite intense competition of other networks at the time "Live and Help Live" was presented.

In the 1954 season plans are developing for a large unit which will concern the growth and development of the human being. Anatomic and physiologic development will be considered as well as emotional and intellectual growth. This will be a joint presentation from the basic science and clinical departments working together. Dr. Louis A. M. Krause will present a program on geriatrics. At Christmas time an appropriate Christmas program will again be presented. In fact, the number of requests received from the public for programs almost equals the amount of time allocated for their presentation. "Live and Help Live" therefore looks forward to a new year of continued service. Undoubtedly the coming year's programs will reflect the experience and improving craftsmanship of those physicians who have so wholeheartedly cooperated in the development of this weekly series.

Programs given during the year 1953–54 are listed as follows. All programs were under the direction of Anne Holland.

| | | |
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| Oct. 5 | Opening program with word of welcome by Dr. H. C. Byrd, President of the University, the Deans of the Baltimore Schools, Mr. George H. Buck, Director of the University Hospital and Dr. Dietrich C. Smith, Vice-Chairman of the Post Graduate Committee. | |
| | Scientific Presentation: *Ragweed hay Fever* | Dr. Howard M. Bubert |
| Oct. 12 | *The story of cleft lip and cleft palate* | Dr. Edward Kitlowski |
| Oct. 19 | *How we develop from a single cell* (Embryology) | Dr. Frank H. J. Figge |
| Oct. 26 thru Nov. 9 | *The heart—what is it—how does it work?* | Dr. D. C. Smith<br>Dr. Vernon Krahl |
| Nov. 2 | *The rheumatic heart* | Dr. William S. Love, Jr. |
| Nov. 9 | *The rheumatic heart—its treatment* | |
| | a. The Failing Heart | Dr. William S. Love, Jr. |
| | b. Surgery of the Rheumatic Heart | Dr. R. Adams Cowley |
| | c. Prevention and control of Rheumatic Heart Disease | Dr. C. Edward Leach |
| Nov. 16 | *Pneumonia* | Dr. Wetherbee Fort |
| Nov. 23 | *Peptic ulcer* | Dr. Carl Ebeling<br>Dr. John M. Dennis |
| Dec. 7 | *Legal medicine* | Dr. Russell S. Fisher |
| Dec. 14 | *Cancer* | Dr. Edwin H. Stewart, Jr.<br>Dr. Louis E. Goodman<br>Dr. Arthur Siwinski<br>Dr. C. N. Davidson |
| Dec. 21 | *Medicine and the nativity* | Dr. Louis A. M. Krause<br>Students from the Schools of Medicine and Nursing<br>The Nurses' Choir of the University Hospital<br>Dr. John A. Wagner, *Narrator* |
| Dec. 28 | *"Don't let tragedy spoil your holiday"*<br>Safety Program sponsored by Anne Holland and WBAL including members of the State Police, Physicians and Safety Experts. | |
| *1954* | | |
| Jan. 4 | *How we grow from a single cell* (Oct. 19 telecast continued) | Dr. Frank H. J. Figge and Staff |
| Jan. 11 | *The care of the normal skin* | Dr. Harry M. Robinson, Jr. |
| Jan. 18 | *Bronchial asthma* | Dr. Howard M. Bubert |
| Jan. 25 | *Resuscitation of the newborn* | Dr. Louis H. Douglass |
| Feb. 1 | *Surgical nursing* | Nursing School of the University of Maryland including Dean Florence Gipe, Miss Kathryn Williams, Miss Rita Malek, Miss Ann Lee DeHaven |
| Feb. 8 | *Lead poisoning in children* | Dr. J. E. Bradley and Staff |
| Feb. 15 | *Hematology* | Dr. Milton S. Sacks and Staff |
| Feb. 22 | *Resistance to infection* | Dr. Merrill S. Snyder<br>Dr. Edward Steers<br>Dr. Andrew G. Smith |
| Mar. 1 | *Congenital malformations of the kidney* | Dr. Albert E. Goldstein |
| Mar. 8 | *Living with diabetes* | Dr. J. S. Eastland |

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|---|---|---|
| Mar. 22 | *Management of head injuries* | Dr. R. K. Thompson<br>Dr. W. S. Mosberg |
| Mar. 29 | *Pelvic inflammation* | Dr. Everett S. Diggs<br>Dr. E. I. Cornbrooks<br>Dr. Theodore Kardash<br>Mr. Thomas Stebbins |
| Apr. 5 | *Program for the hard of hearing* | Dr. James Gerlach |
| Apr. 12 | *The doctor and the bible* (A Medical-Religious Symposium) | Dr. Louis A. M. Krause<br>Rev. William Hill<br>Rev. Herman Schulz<br>Rabbi Israel Goldman |
| Apr. 19 | *The intern is your doctor* | University Hospital House Staff and the Senior Residents |
| Apr. 26 | *The story of belladonna* | Dr. B. F. Allen<br>Dr. Casimir Ichniowski<br>Dr. Frank Slama |
| May 3 | *Summer skin hazards* | Dr. Harry M. Robinson, Sr.<br>Dr. Harry M. Robinson, Jr. |
| May 10 | *The hospital chart* (University Hospital Program) | Mr. George H. Buck and Staff |
| May 17 | *Chemistry on the surface* | Dr. George Hager of the School of Pharmacy |
| May 24 | *Gout and gouty arthritis* | Dr. Leon Kochman |
| | *Closing remarks* (Panel)<br>Anne Holland<br>Judge William Cole, Jr., Chairman of the Board of Regents<br>Acting President T. B. Symons<br>The Deans of the Baltimore Schools<br>Mr. George H. Buck<br>Dr. Howard M. Bubert | |

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## University of Maryland Biological Society

President, *Frederick P. Ferguson* Secretary, *Edward Steers*

## PROGRAM FOR THE ACADEMIC YEAR 1953–54

**November 10, 1953**—The Annual Business Meeting, Bressler Library, Chief items of business transacted included:

1. Election of the following officers for the year 1953–54:
   President—Frederick P. Ferguson
   Secretary—Edward Steers
   Treasurer—Donald E. Shay
   Counsellor—Raymond M. Burgison
2. Election of twenty-one (21) nominees to associate membership and one (1) nominee for honorary membership.

At the conclusion of the business meeting, Dr. John C. Krantz, Jr., Professor of Pharmacology, School of Medicine, University of Maryland, presented an interesting account of "Some Impressions of the XIX International Physiological Congress Held in Montreal in September 1953".

**December 16, 1953,** Bressler Library. "Report on the First Teaching Institute in Physiology, Biochemistry and Pharmacology" by Dr. William R. Amberson, Professor of Physiology, School of Medicine, University of Marvland.

**January 27, 1954,** Bressler Library. "Intermediary Metabolism of Purines with Specific Reference to a Chloramphenicol Resistant Strain of *Micrococcus pyogenes*, var. *aureus*", Robert C. Wood and Edward Steers, Department of Bacteriology, School of Medicine, University of Maryland.

**February 24, 1954,** Bressler Library. "Some Problems in the Evaluation of Blood Pressure Depressant Drugs", Raymond M. Burgison, Department of Pharmacology, School of Medicine, University of Maryland.

**March 31, 1954,** Bressler Library. "Treatment of Coeliac Disease with Powdered Duodenum", Gordon E. Gibbs, Department of Pediatrics, School of Medicine, University of Maryland. "Enzyme Studies of a Chloramphenicol Resistant Strain of *Escherichia coli*", Mary V. Reed and Edward Steers, Department of Bacteriology, School of Medicine, University of Maryland.

**May 12, 1954**—Dinner Meeting, Marty's Restaurant. "Humoral Control of Red Blood Cell Formation", Dr. Wilson C. Grant, Head, Department of Physiology, Lederle Laboratories Division, American Cyanamid Company, Pearl River, New York.

## THE PURINE METABOLISM OF A CHLORAMPHENICOL-RESISTANT STRAIN OF *MICROCOCCUS PYOGENES*, VAR. *AUREUS*

ROBERT C. WOOD AND EDWARD STEERS
DEPARTMENT OF BACTERIOLOGY

*Abstract*

In order to study the mode of action of the antibiotic chloramphenicol, a strain of *Micrococcus pyogenes* var. *aureus* was developed which is resistant to the highest soluble concentration of the drug.

The chloramphenicol-resistant strain, unlike the parent sensitive strain, requires adenine plus another purine for growth. A comparative study of the purine metabolism of the parent strain and of the resistant strain may lead to an elucidation of the mechanism by which resistance to chloramphenicol is achieved.

Growth experiments were made using the postulated precursors of the purines (glycine, formate, bicarbonate, ammonium ion, 4-amino-5-imidazolecarboxamide and the B vitamins) and isotopic studies using adenine-8-$C^{14}$ and guanine-8-$C^{14}$. Such experiments demonstrated that the resistant strain cannot utilize these simple precursors for growth nor interconvert adenine and guanine. The sensitive strain, however, utilizes simple precursors for purines (it has no purine requirement) and can interconvert adenine and guanine, guanine preferentially going to adenine.

Isoguanine, 2,6-diaminopurine (DAP), hypoxanthine, and xanthine have been proposed by Brown as possible intermediates in adenine-guanine interconversion. Our studies show isoguanine to be incapable of producing growth, and thus an unlikely intermediate. DAP is inhibitory when used in combination with adenine and guanine. (Our studies demonstrate this inhibition to be caused by a specific blocking of the condensation of guanine with ribose to form guanosine.) DAP-2-$C^{14}$ is not incorporated into polynucleotide purines in the resistant strain and is only incorporated 12 to 13 per cent into PNA adenine and guanine in the sensitive strain. We, therefore, feel that DAP is not an intermediate in adenine-guanine interconversion. Hypoxanthine and xanthine both support growth when used with adenine and are the most likely intermediates in this interconversion.

Comparative growth studies with the purine bases, their ribosides and their ribotides indicate that the free bases are unlikely intermediates. The weight of evidence supports the view that the ribotides are the natural intermediates.

Further experiments are in progress to determine whether substances other than the proposed precursors of the purines may be capable of substituting for the purine requirement in the resistant strain. Preliminary data suggest that we may indeed be dealing with a strain which has different elementary purine precursors than those demonstrated in other organisms.

## VASODEPRESSOR EFFECTS OF SOME 8-SUBSTITUTED THEOPHYLLINE DERIVATIVES

RAYMOND M. BURGISON

*Abstract*

A series of theophylline derivatives with substituents in the 8-position of the molecule (alkyl, aryl, aryalkyl, heterocyclic, amino, alkylamino, aralkylamino, arylamino, carboxy and a number of miscellaneous derivatives) have been studied, principally as blood pressure depressants. The compounds were screened as vasodepressor agents by intravenous injection into anesthetized dogs. The mean arterial blood pressure was recorded by use of a mercury manometer connected to a carotid artery cannula. Practically all compounds studied caused a fall in blood pressure, varying from a few millimeters Hg to as much as 50 per cent of the original mean arterial pressure. The effect of some compounds was transient, while others caused a prolonged fall in blood pressure from which the animal did not recover. In general (with certain important exceptions) hypotensive potency among the compounds studied varied according to the following structural types, arranged in order of decreasing activity: Aryalkyl > Aryl > Heterocyclic > Alkyl > Arylamino > Carboxy > Amino. When the effectiveness of a compound as a hypotensive agent was established by the intravenous route, it was administered intragastrically in order to determine if the compound was active by gut. Most of the compounds studied were not apparently absorbed by gut. However, a few showed good activity by this route of administration. Several compounds were also studied when administered orally, using an arterial puncture technic, over a period of time in trained unanesthetized dogs. The pharmacology of several theophylline derivatives was studied in detail.

## POWDERED DUODENUM IN TREATMENT OF CELIAC DISEASE

GORDON E. GIBBS, M.D.

Celiac disease is a condition characterized by impaired intestinal absorption of carbohydrate and fat. It is thus distinct from cystic fibrosis of the pancreas in which the nutritional defect is lack of pancreatic digestive juice. Although dietary management of celiac disease has been fairly satisfactory, the fundamental pathogenesis remains obscure.

Two years ago the idea occurred to the writer that the disease might involve deficiency of some specific substance which could be provided by administration of normal intestinal mucosa. For testing this possibility, a preparation of powdered raw duodenum was available (Viodenum). This preparation consists largely of protein and is rich in phosphatase and enterokinase. Also it presumably contains other physiologically active principles. It has been given to 10 infants with celiac disease in the present study. In each case there has been an observed control period prior to a period of trial of the duodenal powder. In most instances the routine dietary celiac regime was withheld or given in only partial form during these experimental periods.

In all cases the infants have done well with respect to weight gain and improvement of the stools. The dosage given, namely 0.5 to 2.0 gm. accompanying each feeding, has been well tolerated by the patients and has been at least compatible with prompt recovery. In all cases except one, the recovery persisted after discontinuation of the medication. The one case in which relapse occurred provided a valuable opportunity to demonstrate a second response in the same patient.

In view of the possibility of coincidences, and the presence of various confusing details, any conclusion that the recoveries were definitely accelerated by the duodenal powder would be premature at this time. Further studies have been initiated and more strictly controlled data will be obtained as rapidly as suitable cases become available.

## ENZYME STUDIES OF A CHLORAMPHENICOL RESISTANT STRAIN OF *ESCHERICHIA COLI*

MARY V. REED AND EDWARD STEERS

DEPARTMENT OF BACTERIOLOGY

### *Abstract*

Manometric techniques were used to compare the formic dehydrogenase and formic hydrogenlyase activities of resting cells of a chloramphenicol-sensitive strain and a chloramphenicol-resistant strain of *Escherichia coli*. Maximum activity was obtained with fresh cell suspensions at pH 6.0.

Although the formic hydrogenlyase activity of the two strains was almost the same, the formic dehydrogenase activity of the resistant strain was much greater than that of the sensitive strain. In fact, the resistant strain produced more gas in the presence of methylene blue than in its absence.

Chloramphenicol could not be shown to substitute for methylene blue as an hydrogen acceptor. The antibiotic slightly depressed gas production by the sensitive strain and greatly depressed gas production by the resistant strain. The depressing effect of chloramphenicol on gas production by the resistant strain was immediately relieved by the addition of methylene blue.

An attempt was made to correlate dehydrogenase activity with aryl amine formation at pH 7.42. Thunberg techniques were used to screen various compounds as substrates for dehydrogenase activity. The greatest dehydrogenase activity and aryl amine formation occurred in the presence of formate, glucose, lactate, and succinate. Although no dehydrogenase activity could be demonstrated in the presence of d-ketoglutarate, fumarate, malate, and cis-aconitate, these compounds were utilized by both strains in the formation of aryl amine. Acetate did not play a role in dehydrogenase activity but the sensitive strain produced aryl amine in its presence.

From the results obtained it is postulated that the resistant cells are deficient in natural hydrogen acceptors and/or an electron-carrier.

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The names listed above are officers for the term beginning July 1, 1953 and ending June 30, 1954.

## SCHOOL PHOTOS NEEDED

*Alumni Requested to Search Files and Old Albums for U. of M. Pictures*

In the months to come the Bulletin is planning a photographic survey of the University of Maryland as it was and as it is, including photographs of professors, buildings now no longer in existence and some of the new additions to the School of Medicine. If you, as an Alumnus, have photographs in your possession which might contribute to this project, the Bulletin would be very grateful for their loan (or their gift), all prints to be returned if desired.

In submitting these pictures please give an appropriate legend on the reverse side or on a small slip of paper attached to the photograph. Consider personalities and buildings of utmost importance. Please think of the value of the photograph or reproduction in making the selection of the ones you plan to loan us. These pictures when printed will become a permanent record of the School of Medicine and no doubt will be of great historic value. All photographs should be mailed to the Bulletin of the School of Medicine, University of Maryland, Lombard and Greene Streets, Baltimore 1, Maryland.

## ALUMNI ASSOCIATION PLAN DINNER AT A.M.A. CLINICAL SESSION

*Alumni Urged to Register at A.M.A. Miami Session November 29–December 2*

The Medical Alumni Association has announced the organization of a University of Maryland Alumni dinner incident to the American Medical Association Clinical Meeting to be held in Miami, Florida November 29 to December 2. Alumni are urged to register at the University of Maryland Alumni table and to secure tickets for the dinner at the time of registration. The Chairman of the dinner is Dr. Daniel O. Hammond, class of 1942, who practices in Miami, Florida.

## OBITUARIES

### Dr. Frank W. Grove

Dr. Frank W. Grove, class of 1880, died at his home in Luray, Virginia on November 6, 1953. Dr. Grove was 98. For a number of years Dr. Grove was Medical Examiner of Page County, Virginia.

### Dr. Robert W. Johnson

Dr. Robert W. Johnson, class of 1912, died in the Aiken County Hospital, Aiken, South Carolina on March 16, 1954. Dr. Johnson was 65.

Following his graduation from the School of Medicine he became associated with the teaching faculty and was also a member of the South Baltimore General Hospital staff. After practicing medicine for about 30 years in Baltimore he moved to Augusta, Georgia where he lived for the past 10 years. Two of Dr. Johnson's brothers are physicians: Dr. H. M. Johnson of Columbia, South Carolina and Dr. W. R. Johnson, a twin brother, of Baltimore.

### Dr. Lee Wallace Rork

Dr. Lee W. Rork, class of 1896, died at his home in Hastings, Nebraska on December 23, 1952. After his graduation Dr. Rork began his career as a general practitioner, most of his time being spent in Oxford, Nebraska. In 1923 he moved to Hastings and after graduate study limited his practice to roentgenology. He was for many years a member of the Hastings State Hospital staff, the American College of Radiology and the Radiological Society of North America.

### Dr. Henry Lyon Sinskey

Dr. Henry L. Sinskey, class of 1908, and former chief of the nose and throat clinics at the Sinai and University Hospitals, died at Union Memorial Hospital on September 15, 1953 at the age of 66. Dr. Sinskey was a native of Baltimore, a graduate of Baltimore City College and at one time served as coroner of Baltimore City.

### Dr. Wilbur F. Skillman

Dr. Wilbur F. Skillman, class of 1900, died on Friday, November 6, 1953, at his home, 6 East Biddle Street in Baltimore after an illness of several years.

The son of the late George and Mary Elizabeth Skillman, he was born in Baltimore and after preliminary education in the local schools was graduated from the University of Maryland School of Medicine. He then practiced in Baltimore for the remainder of his life. Dr. Skillman was a member of the staffs of the Lutheran Hospital and the Union Memorial Hospital. He was also a consultant at the Southern Hospital and Home. Dr. Skillman's chief interest in his later years was his Medical Directorship of the Masonic Home at Cockeysville, Maryland.

## Dr. Francis M. Walker

Dr. Francis M. Walker, P & S, class of 1906, died at his home in Rochester, New York on July 1, 1953 of coronary occlusion.

Dr. Walker was for many years a general practitioner and surgeon in Rochester, New York.

One of the 8 honor men of his class, he served a term as resident physician at the Baltimore City Hospitals.

## Dr. Ernest R. Zemp

Dr. Ernest R. Zemp, P & S, class of 1894, died at the Fort Sanders Hospital in Knoxville, Tennessee on February 7, 1954. Dr. Zemp was 82.

He was born in Camden, South Carolina in 1871, the son of Dr. and Mrs. Francis L. Zemp. He received a Bachelor of Science degree at The Citadel in 1890. After his graduation from the College of Physicians and Surgeons he returned to Knoxville remaining in active practice for over 60 years.

A leader in local, state and regional medical associations, Dr. Zemp was also sought by other fields and groups as a speaker. He was a past president of Rotary, of the State Medical Association, the East Tennessee Medical Society and the Knoxville Academy of Medicine. He was speaker of the House of Delegates, governing body of the state medical group, and had held that post longer than any other physician. Dr. Zemp was an instructor at the old Lincoln Memorial University Medical School at the beginning of the century.

**Anderson, Charles Rhodes,** Winchester, Va.; class of 1908; aged 75; served during World War I; died, October 24, 1953.

**Bauersfeld, Emil George,** Bethesda, Md.; B.M.C., class of 1911; aged 64; died, December 13, 1953.

**Bayer, Ira Eugene, Jr.,** Baltimore, Md.; class of 1934; aged 47; died recently.

**Fernald, Arthur Blair,** St. Petersburg, Fla.; B.M.C., class of 1898; aged 82; died, October 31, 1953, of coronary thrombosis and hypertension.

**Girouard, Joseph Arthur,** Willimantic, Conn.; B.M.C., class of 1899; aged 78; died, September 29, 1953, of coronary thrombosis.

**Good, Benjamin F.,** Lancaster, Pa.; B.M.C., class of 1900; aged 81; died, October 23, 1953.

**Herr, John Trout,** Landisville, Pa.; class of 1910; aged 67; died, October 11, 1953, of arteriosclerotic heart disease.

**Higgins, Martin Andrew,** Rye, N. H.; B.M.C., class of 1907; aged 69; died, August 18, 1953, of acute coronary occlusion.

**Kenney, Stephen A.,** Valley Falls, R. I.; P & S, class of 1899; aged 77; died, September 14, 1953, of cerebral arteriosclerosis.

**Lawson, Sidney B.,** Logan, W. Va.; B.M.C., class of 1894; aged 86; died, October 9, 1953, of asthma and heart disease.

**Little, Theodore Alvin,** Erie, Pa.; B.M.C., class of 1908; served during World War I; aged 71; died, August 16, 1953, of carcinoma of the prostate.

**McLaughlin, Calvin Sturgeon,** Charlotte, N. C.; class of 1896; aged 83; died, November 15, 1953, of acute myocardial failure.

**McLean, Isaac William,** Charlotte, N. C.; B.M.C., class of 1907; aged 72; died, September 30, 1953, of heart disease.

**McPherson, Samuel Dace,** Durham, N. C.; class of 1903; aged 80; died, September 16, 1953, of arteriosclerosis.

**Meanwell, Walter Ernest,** Madison, Wisc.; B.M.C., class of 1909; aged 69; died, December 3, 1953, of coronary heart disease.

**Miles, Clarence Gardner,** Brockton, Mass.; P & S, class of 1908; aged 73; died, October 3, 1953, of coronary thrombosis and rheumatic heart disease.

**Norman, J. Standing,** Gastonia, N. C.; class of 1909; aged 76; died, October 6, 1953, of carcinomatosis.

**O'Hara, James Francis, Jr.,** Canton, O.; class of 1940; aged 38; died, October 11, 1953.

**Pence, Charles W.,** Philadelphia, Pa.; class of 1894; aged 83; died, October 7, 1953, of coronary thrombosis.

**Pound, John Charles,** Baltimore, Md.; B.M.C., class of 1896; aged 89; died, November 27, 1953, of arteriosclerotic cardiovascular disease.

**Shatto, Arthur Blair,** St. Petersburg, Fla.; P & S, class of 1895; aged 82; died, October 11, 1953, of arteriosclerotic heart disease.

**Tyson, William Elisha Ellicott,** Detroit, Mich.; class of 1905; aged 76; died, June 18, 1953, of myocardial infarction.

**Walker, Francis Milton,** Rochester, N. Y.; P & S, class of 1906; aged 72; died, July 1, 1953, of coronary occlusion.

**Weber, John Joseph,** Pittsburgh, Pa.; class of 1917; aged 11; died, July 29, 1953, of cerebral hemorrhage.

**Williams, John Watkins,** Williamston, N. C.; class of 1906; aged 68; died, May 19, 1953, of uremia and auricular fibrillation.

**Wilson, Harry Edmond,** Port Orchard, Wash.; P & S, class of 1905; aged 74; died, April 4, 1953, of coronary thrombosis.

**Wolfe, Jacob S.,** Bloomfield, N. J.; P & S, class of 1890; aged 87; died, May 17, 1953, of bronchopneumonia.

**Zimmerman, Ira Melvin,** Williamsport, Md.; B.M.C., class of 1913; aged 61; served during World War I; died, March 17, 1953, of injuries received in an automobile accident.

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| Riboflavin | 2.0 mg. |
| Pyridoxine Hydrochloride | 0.5 mg. |
| Nicotinamide | 10.0 mg. |
| Ferrous Gluconate | 5.0 grs. |
| Folic Acid | 0.67 mg. |
| Vitamin $B_{12}$ Activity | 5.0 mcg. |

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*Bulletin* OF
THE SCHOOL OF MEDICINE
*UNIVERSITY OF MARYLAND*

VOLUME 39 *October, 1954* NUMBER 4

## EDITORIAL

### DR. FRED W. RANKIN

The award for the most distinguished graduate of the University of Maryland School of Medicine had to be given posthumously this year. Dr. Rankin, the recipient, died just a few days before the annual meeting of the Alumni Association.

It was my privilege to have known Dr. Rankin in his younger days when he was a house officer at the University Hospital and later when he was in practice in Baltimore. Although our paths diverged after this, our early association was such that I always felt extremely close to him and am conscious of a great sense of loss at his passing.

The latter part of his career is too well known to require any comment from me and it is of his earlier years that I wish to write here.

Fred Rankin's outstanding characteristic was the fact that he was most human. He had a wide variety of interests and an infinite capacity for enjoying life even in its simplest aspects. As a companion his keen sense of humor and his ready wit made him most enjoyable. He had the rare ability to fit himself to his environment and to become an integral part of any company in which he was placed.

While not necessary, these simple remarks would not be complete without reference to his ability in his profession. Very early he demonstrated the manual dexterity which characterized his work throughout his career and it was always an enlightening experience to watch him operate. Also, he developed rapidly in surgical judgment and appeared to have an uncanny knowledge as to when to operate and when not to. His interest in his work and his ability to learn were great and a day seldom passed when he did not increase his knowledge.

So that, even in his formative years Dr. Fred W. Rankin gave ample evidence of those qualities which served to make him the outstanding figure in the surgical world which he became. He fully deserved the honor awarded him by our Alumni Association and the many other honors which he received.

Finally, as one individual, my life was greatly enriched by having known him and I am proud that I was able to call him "Friend".

*Louis H. Douglass, M.D.*

# RECENT ADVANCES IN THERAPY*†

JOHN C. KRANTZ, JR.

The one dominant feature of modern drug therapy is change. During the past decade these changes have occurred with a rapidity that is overwhelming. The magnitude of the mutation is evinced by the statement that 80 per cent of the pharmacologic agents available in the armamentarium of the physician in 1954 were not known a decade ago. Indeed the promise for the future appears equally fruitful. No chemical structure seems to be able to thwart the enterprising synthetic efforts of the organic chemist. From his assembly line new organic chemicals roll off at the prodigious rate of about 10,000 a year. With the indefatigable efforts of the pharmacologist and bacteriologist in screening compounds for therapeutic value, one anticipates the demonstration of the usefulness of many of these agents annually in the treatment of disease. The search is exciting, the struggle intriguing, and the stakes are high.

## THE ANTI-INFECTIVE DRUGS

The first consideration of this discussion will be the status of the anti-infective drugs. Despite the rapid advances in antibiotic therapy, the sulfonamide drugs still occupy an enviable position in the treatment of infectious disease. This is shown by the fact that in 1953 we produced approximately four million pounds of the various sulfonamides. Through an ever-lengthening experience with these compounds it now appears that the triple sulfonamide offers clinical advantages which are not encountered in the administration of a single pyrimidine sulfonamide.

Sulfonamide therapy today may be divided into two segments. One-half of the therapy involves the use of this triple sulfonamide. On the other hand, Gantrisin®, which does not contain the pyrimidine nucleus, comprises 40 per cent of the sulfonamide usage in this country and is rapidly increasing. The remaining 10 per cent is composed of the use of Elkosin® and other sulfonamides. Recently, Gantrisin has been made available as its $N^1$ acetyl derivative. This is insoluble and tasteless. It obviates one of the objections to Gantrisin therapy, namely, the disagreeable bitter taste of the drug. In the body it is broken down into Gantrisin. This acetylation is not the same as occurs in the body. Here acetylation occurs on position $N^4$ and not $N^1$. (Fig. 1.)

The triad of broad spectrum antibiotics of the late forties, namely, Chloromycetin®, Aureomycin® and Terramycin®, has been substantially augmented by Erythromycin® and Carbomycin®. It now becomes crystal clear that many of such agents are available from the metabolic processes of the *Streptomyces*. Thus the problem of the development of resistant strains of pathogenic bacteria becomes ever less important. We are now in a position to view with the unprejudiced vision that only sufficient retrospect can confer, the relative utilitarian value of these drugs as measured against the first generally-used antibiotics, namely, penicillin. Thus in the spectrum of infectious diseases responding to antibiotic therapy the following compilation of data is revealing:

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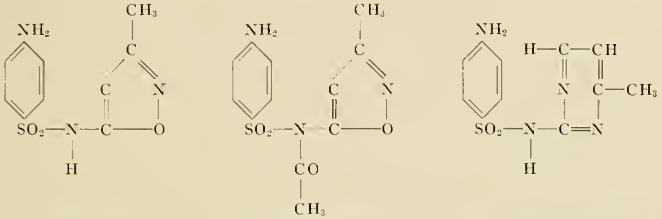
* Presented before the Sixth Annual Scientific Assembly of the American Academy of General Practice, Cleveland, March 25, 1954.

† From The Department of Pharmacology, University of Maryland School of Medicine, Baltimore.

| | |
|---|---|
| Penicillin | 71.8 per cent |
| Broad spectrum antibiotics | 7.4 per cent |
| Streptomycin | 7.1 per cent |
| Sulfonamides | 13.7 per cent |

These data are shown graphically in figure 2.

This information is easily comprehensible when one considers that the penicillin spectrum includes syphilis (240,000 new cases each year), gonorrhea, streptococcic and pneumococcic pneumonias, staphylococcic and streptococcic bacteremias, meningitis and many other commonly-occurring infections. The lack of toxicity of penicillin, its inexpensiveness and its availability in many dosage forms confer upon it a unique stature among the antibiotics. Indeed the prediction of its discoverer, the Nobel Laureate Sir Alexander Fleming, is being continuously unfolded on the



Gantrisin

N′ acetyl Gantrisin

Typical pyrimidine sulfonamide, sulfamerazine

FIG. 1

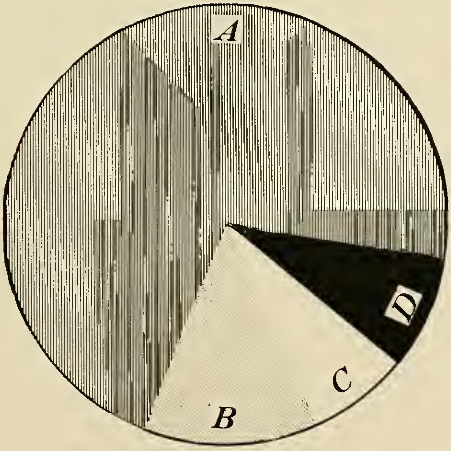


FIG. 2. Spectrum of common infections. *A*—Penicillin, antibiotic of first choice, 40,785,000. *B*—Sulfonamides, drug of first choice, 7,822,000. *C*—Streptomycin antibiotic of first choice, 4,000,000. *D*—Broad-spectrum antibiotics, antibiotic of first choice, 4,200,000.

antibiotic horizon. He prophesized, "It is doubtful if an antibiotic will ever be found that will be more effective than penicillin against those infections caused by penicillin-susceptible organisms."

Let us examine the record to determine if these postulations are substantiated by practice. In 1950 we used one hundred and thirty-eight trillion units of penicillin; by 1952 this had increased to three hundred and sixty trillion units—nearly a three-old increase in two years.

### HYPERTENSION

In recent years the armamentarium in the treatment of hypertension has been enriched by the development and clinical use of Hexamethonium Chloride (ganglionic blocking agent); (2) Apresoline® (depressant vasoconstrictor center), and (3) the hydrogenated ergot alkaloids (depressant to vasoconstrictor center with some peripheral adrenergic blockade). Each of these agents is useful in certain cases of hypertension. Each is fraught with untoward side reactions. This is especially objectionable in mild hypertensives of neurogenic etiology where the hypotensive drugs elicit more severe untoward symptoms than the disease. It is therefore with enthusiasm one welcomes the advent of Rauwolfia serpentina into occidental materia medica for its action in hypertension.

Rauwolfia serpentina is a tropical shrub growing widely in India. For centuries the root of the plant has been used in Indian medicine. The powdered root mixed with pepper was employed in the treatment of the insane. Rauwolfia elicits a sedative effect. Sen and Bose (1931) recorded that alkaloids obtained from rauwolfia root elicited hypotensive effects. Real interest in rauwolfia in occidental medicine stems from the work of Vakil (1949) who used the extract in the treatment of hypertension.

Several alkaloids have been isolated from Rauwolfia serpentina. These are *serpentine*, *serpentinine*, *ajmaline*, *ajmalinine*, *reserpine* and *serpogenine*. Reserpine is the most potent agent so far isolated and probably accounts for much of the activity of rauwolfia. The effect of reserpine can be detected in experimental animals even at a dose of 1 mcg./kg./day given orally for five days. Reserpine is an ester-alkaloid like the hypotensive veratrum alkaloids. Degradation studies produced evidence to show that reserpine contains the pentacyclic nucleus which is present in the alkaloid yohimbine.

At present the most satisfactory therapeutic results are obtained by the use of the extract of rauwolfia.

*Mechanism of action of rauwolfia.* The principal pharmacologic responses of Rauwolfia serpentina are *hypotension*, *bradycardia*, and *sedation*. In contrast to most other known agents producing such effects, rauwolfia in clinical doses exhibits a latent period of several days before these changes become apparent. It is therefore necessary to make a distinction between the immediate effects of very large doses of rauwolfia and the results from clinical doses after the latent period.

The cause of the delayed action is not known. There is, however, reason to believe that the results of acute experiments with massive doses involve mechanisms foreign to the clinical effects.

The bradycardia elicited by rauwolfia is of vagal origin and is readily blocked by atropine. The apparent increase in vagal tone may be merely a resultant of adrenolytic effects upon the sympathetic accelerator fibers to the heart. The principal electrocardiographic pattern change in associated with the bradycardia such as an increase in the duration of the S-T segment.

It is likely that the sedative effect produced by rauwolfia contributes to its efficacy in the treatment of hypertension. Wilkins and Judson (1953) found rauwolfia useful in the treatment of less severe and labile types of hypertension. From 30 to 50 per cent of these patients reacted favorably subjectively and objectively to the treatment. Hypertension of nervous origin with tachycardia responded best to the action of rauwolfia. Severe hypertensives with renal insufficiency did not respond well to the treatment with rauwolfia alone. However, the sedative action of the drug appeared beneficial.

The drug reduces systolic and diastolic blood pressures in those individuals where it is effective. In most cases this is accompanied by a bradycardia. The patients frequently experience a sense of well-being and relaxation.

The side effects of rauwolfia so far encountered are few and not severe. Subjective tremors have been experienced in patients taking large doses. These disappeared upon cessation of treatment. Others have experienced an abundant watery secretion of the bronchial tubes acting similarly to potassium iodide. Occasionally patients have reported nasal stuffiness, diarrhea, drowsiness, disturbed sleep, and vertigo. Rauwolfia treatment should be enjoined with caution in patients with cardiac hypertrophy.

Rauwolfia is administered in tablets of 2 mg. of the alkaloidal extract. Two tablets are given daily until the desired effect is achieved and then 1 tablet daily. Larger doses are well tolerated in many hypertensives. The drug is available under the names Raudixin® and Rauwiloid®. Rauwolfia is also available with veratrum alkaloids for more persistent types of hypertension.

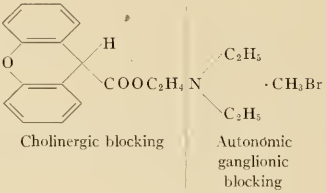
One welcomes rauwolfia to the armamentarium. As its mechanism of action is more definitely elucidated it is hoped that the early clinical claims will be further substantiated. Indeed a new interest has been awakened in the chemistry of the yohimbine nucleus, which structure appears to be present in reserpine. The future of this new attack on hypertension looks hopeful.

### PEPTIC ULCER

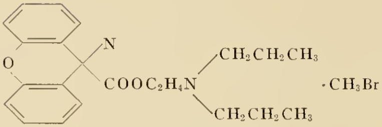
In the therapy of peptic ulcer, Pro-Banthine® Bromide has taken its place along side of Banthine® as an anticholinergic and autonomic ganglionic blocking agent. Pro-Banthine is a more potent cholinergic blocking agent than Banthine. The chief advantages of Pro-Banthine over Banthine appear to be first, that it is two to five times more potent than Banthine, and second, the incidence of untoward reactions is smaller. The two compounds differ structurally. Pro-Banthine contains two propyl groups attached to the quaternary nitrogen atom instead of two ethyl groups as is present in Banthine. The indications of the two drugs are the same.

The recommended dosage schedule of Pro-Banthine is 15 mg. (one tablet) with meals and 30 mg. at bedtime. Reports of the clinical use of Pro-Banthine are equally as favorable as those with Banthine, and the regimen is apparently fraught with fewer disturbing side reactions.

There are now available at least four of these anticholinergic ganglionic blocking



Banthine



Probanthine

Fig. 3

spasmolytics, namely, Banthine, Pro-Banthine, Antrenyl®, and Prantal®. It appears that in certain cases of hypermotility of the gastrointestinal tract any one of them may be superior to atropine. However, the vast majority of the cases are better controlled with fewer untoward symptoms with atropine. Further, of the four compounds it is difficult to point to a drug of choice. It is possible that further extensive trial will determine this. However, it occurs to this observer that their choice will remain a matter of selectively fitting the drug to the patient's needs with minimal side effects. Also, that certain patients will be benefitted more with one spasmolytic and others with another. Indeed this seems to be established with the long used spasmolytics which do not produce ganglionic blocking such as atropine, Syntropan and Trasentin.

### HEADACHE

There is no more baffling problem to the physician nor a more disconcerting one to the patient than a headache. It is estimated that last year the American public suffered with a half-billion headaches. To combat this they used 18 tons of aspirin daily along with many additional tons daily of patented headache powders. Interesting data are now available to use in the treatment of the hypertensive headache.

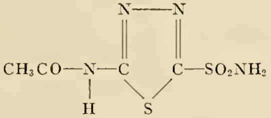
Caffeine has been used widely in the treatment of headache. It is a frequent constituent of the generally-used headache remedies. Moyer et al. (1952) placed its therapeutic use in hypertensive headaches on a sound basis. Caffeine was shown to decrease the arterial and postarteriolar vascular tension of cerebral vessels and through this mechanism relieve hypertensive headaches. A decrease in cerebral blood flow was demonstrated. This was followed by a reduction in cerebrospinal fluid pressure. The fall in cerebrospinal fluid pressure is secondary to the decrease in cerebral blood flow. Aminophylline shares this action with caffeine. Moyer and his associates found aminophylline to be the more effective of the two drugs in the relief of hypertensive headache.

## DIURESIS AND CARBONIC ANHYDRASE

Carbonic anhydrase is an enzyme which converts carbon dioxide entering the blood from the tissues into carbonic acid. It was established many years ago that the administration of sulfanilamide caused the inhibition of carbonic anhydrase. The inhibition of carbonic anhydrase resulted in diuresis. Thus investigators sought to prepare a potent carbonic anhydrase inhibitor with a low degree of toxicity. Roblin et al. (1950) prepared a series of heterocyclic sulfonamides; among these 2-acetylamino-1,3,4-thiadiazole-5-sulfonamide appeared to meet these requirements. This sulfonamide is available under the name of *Acetazoleamide*® or *Diamox*®.

Diamox is different in chemical constitution and pharmacologic activity from the generally employed bacteriostatic sulfonamides. It is rapidly absorbed from the gastrointestinal tract and almost completely excreted unchanged in the urine within 24 hours. Its principal site of action appears to be in the renal tubules where carbonic anhydrase is inhibited. Thus there is specific interference with the production of hydrogen and bicarbonate ions. The impeding of this acidification process which conserves base, results in a copious diuresis of bicarbonate, sodium potassium ions and water. Thus one observes that Diamox functions as a diuretic through an entirely different mechanism than do the classical diuretics. Since Diamox produces a loss of sodium without a concomitant loss of chloride its action is synergistic with that of the organic mercurials which produce chloruesis. On the other hand, the effect of Diamox is partially blocked by the acidifying diuretic ammonium chloride.

Diamox is used to produce diuresis in edema of cardiac origin. It is given orally once a day in 250 mg. tablets. The preliminary studies using this diuretic, which have come to the attention of this observer, indicate that it is usually effective. Its side reactions are few. One welcomes to the materia medica this new diuretic whose action represents such a wide departure from that of the mercurial diuretics.



Diamox

2-acetylamino-1,3,4 thiadiazole 5-sulfonamide

FIG. 4

$$CO_2 + H_2O \xrightarrow{\text{Carbonic anhydrase}} \overset{\xleftarrow{\text{Site of diamox action}}}{H_2CO_3}$$

$$H_2CO_3 \rightleftarrows H^+ + HCO_3$$

$H^+$ ions replace $Na^+$ ions of glomerular filtrates as shown

$$Na_2HPO_4 + H^+ \rightarrow NaH_2PO_4 + Na^+$$

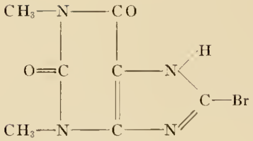
$Na^+$ returns to blood

$K^+$ and $H^+$ ions compete in tubules for excretion

Hence a diminution of $H^+$ ions increases $K^+$ ion excretion

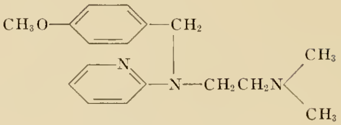
FIG. 5

5 parts



8-bromotheophylline

3 parts



Antihistaminic neoantergan

Neobromth

80 mg. tablets (2 tablets morning and evening).

FIG. 6

### PREMENSTRUAL TENSION

The approach of the menstrual cycle frequently produces symptoms of discomfort. These may be grouped under the heading of premenstrual tension. Bickers and Woods (1951) state, "The bleeding phase of the menstrual cycle rarely appears unannounced." Signs and symptoms which herald its approach are experienced by many women. The principal symptoms include nervousness, irritability, insomnia, backache, headache, breast tenderness, abdominal fullness, thirst, and mild antisocial behavior. The symptoms disappear usually with the beginning of menstrual flow.

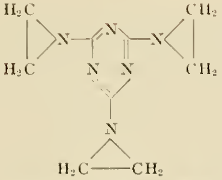
It is believed that altered hormonal balance of the premenstrual period stimulates increased production of the antidiuretic hormone (ADH). Thus salt and water are retained by the tissues. There appears to be general agreement that abnormal water storage in the tissues is the most important incriminating facet of the premenstrual syndrome. Weight gain and symptoms are often concomitant. The weight gains may vary from a few to several pounds.

It follows from the foregoing discussion that drugs valuable in combatting the premenstrual edema would be useful in the treatment of premenstrual tension. For years the acidotic diuretic ammonium chloride has been used for this purpose. Bickers (1951) observed the use of pyrilamine (antihistaminic) combined with 8-bromotheophylline was useful in relieving premenstrual tension. Animal experiments and clinical trials showed that the 8-bromotheophylline portion of the compound was responsible for much of its therapeutic efficacy. It is consistently capable of producing diuresis in the presence of Pitressin® in animals and man.

The theophylline derivative is available as a mixture consisting of 2-amino-2-methyl-1-propanol 8-bromotheophyllinate and pyrilamine maleate in a 5:3 ratio. The mixture is called NeoBromth®. Tablets are available containing 80 mg. of the mixture. The recommended dosage is two tablets morning and evening daily five to seven days prior to menstruation. With this treatment Bickers (1952) found in an adequate series of patients an average premenstrual weight gain of 1.6 pounds compared with 4.2 pounds prior to treatment. In the main, symptoms were mitigated when effective diuresis resulted from treatment.

### THERAPY IN NEOPLASTIC DISEASE

*Triethylene melamine*, (*TEM*). This substance was synthesized in an effort to prepare a more satisfactory agent than nitrogen mustard in the treatment of neoplastic diseases. Its structure is shown in the following formula:



Triethylene melamine (TEM)

5 mg. tablets; (5 mg. daily dose).

Fig. 7

TEM is related to nitrogen mustard when it is transformed into the ethyleneimine rings which are considered the active cytotoxic groups. It is recommended in the treatment of lymphomas, chronic myelogenous leukemia, chronic lymphatic leukemia, Hodgkin's disease, malignant lymphoma, and certain other malignancies. It is active orally as well as parenterally. Nausea and anorexia are common untoward side-effects. Emesis is not frequently encountered.

Intravenously the dose of TEM is 0.05 mg. per kilogram, never to exceed 5 mg. Doses of 0.25 mg. per kilogram intravenously are fatal in man. The oral dose is 0.1 to 0.3 mg. per kilogram. TEM is available in 5 mg. tablets. The maximal oral daily dose usually never exceeds 5 mg.

The major hematologic toxic effects of TEM are similar to those elicited by Mechlorethamine, namely; leukopenia, thrombocytopenia, and less frequently anemia.

The use of TEM in neoplastic disease is discussed by Rundles and Barton (1952). Many remissions in malignant lymphoma have been dramatic under TEM therapy. Results in chronic lymphocytic leukemia have also been encouraging. It will take many years to completely assess the place warranted by TEM in the therapy of neoplastic disease. At present it appears to offer distinct advantages over nitrogen mustard.

Other substances of this character will be forthcoming. Preliminary reports on the compound Triethylene Thiophosphoramide in chronic leukemias are promising. The all too dark horizon in neoplastic disease therapy at long last shows some scintillations.

## FUTURE OUTLOOK

Standing in the shadows of the great pyramids the youthful general Napoleon Bonaparte addressed his soldiers, saying, "Forty centuries are looking down on you." It has been just 100 years since the death of the first pharmacologist, Francois Magendie. Today 10 decades of the ever-lengthening past are looking down on us. Great strides have been made, the azure dawn of the conquest of the infectious diseases is upon us. The ever-approaching future sounds no uncertain challenge. The more difficult, the more perplexing, the more baffling, multifaceted degenerative diseases must also yield to the united charge of the chemist, pharmacologist, and physician. For the clarion call of the people whom we serve echoes ever more poignantly—you have put more years into the life—now your task is to put more life into the ever-increasing years.

# HYPOPARATHYROIDISM WITH CEREBRAL CALCIFICATION:*, †
## I. REPORT OF A CASE

JOHN A. WAGNER, M.D., URSULA T. SLAGER, M.D.,‡ AND LEONARD TUCKER, M.D.§

### INTRODUCTION

Although 30 cases of hypoparathyroidism with bilateral cerebral calcification have been reported, only 4 of these are subsequent to thyroidectomy (1–4). These, together with 34 cases of a more obscure etiology fall histologically into a group of "idiopathic", non-arteriosclerotic, cerebral, vascular calcifications which have been called Fahr's Disease (5), first described by Pick in 1902 (6). Pick believed the tetany to be a result of the cerebral lesion. Since then the causal relationship has been disputed, but currently most authors seem to agree that the cerebral lesions are secondary to hypoparathyroidism (4, 7). As the occurrence of cerebral calcification in parathyreoprivic tetany following thyroidectomy seemed to strengthen this view, this, a fifth case, was believed to be of sufficient interest to be reported in detail.

### CASE REPORT

F. W., a 53 year old white female was admitted to the West Baltimore General Hospital,¶ Baltimore, Maryland on March 10, 1949 with the chief complaint of pain in her fingers of 4 weeks' duration.

*Past History:* There had been a mass in her neck since the age of 10, for which, at the age of 13 or 14, a right thyroid lobectomy was done at the Post Hospital,‖ Fort Howard, Maryland. In 1923 she suffered cranio-cerebral trauma in a riding accident and was unconscious for 15 minutes. In March 1928 fainting spells commenced, occurring more frequently around the time of her menstrual periods. These brought her to the Johns Hopkins Hospital dispensary in September 1928. A gynecologicaly examination revealed nothing of significance. The fainting spells were described as starting with an aura of "feeling a quiver all over", followed by rigidity of the extremities, foaming at the mouth, and unconsciousness. There were no tremors, no urinary or fecal incontinence, and no tongue biting. After the attack, she remained unconscious for some minutes, and did not remember it. A diagnosis of grand mal epilepsy with a possible hysterical component was made. No skull roentgenograms were taken. Examination at the thyroid clinic (for nervousness) disclosed a firm, non-nodular left thyroid lobe and a basal metabolic rate of plus 5 per cent.

In 1929 a subtotal left thyroid lobectomy was performed at The Hospital for the Women of Maryland for this recurrent non-toxic nodular goitre. The surgeon described a grossly normal right thyroid lobe, and the pathologist reported a colloid goitre of the left lobe. No mention of parathyroid tissue was made.

A succession of several convulsive attacks occurred shortly thereafter while traveling in Canada, and an exploratory craniotomy in the right parietal region was performed at Saint Boniface's Hospital in Winnipeg, Canada.** She was told a "clot on her brain" was found. Her convulsions were said to have decreased in frequency following operation. In November 1941 she had a convulsion and when brought by the police to the Johns Hopkins Hospital Accident Room she stated that her attacks had occurred for the past 9 years on the average of twice a year. In July 1942 a cervical dilatation and uterine curettage, and in January 1943 a hysterectomy was performed for menometrorrhagia at The Hospital for the Women of Maryland. No mention is made of her convulsions.

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¶ Now the Lutheran Hospital of Maryland.

‖ Not the Veterans' Administration Hospital.

** No records are available of this hospitalization.

In 1947 she was treated at The Sinai Hospital of Baltimore for bronchopneumonia. An electrocardiogram taken during this admission showed a prolonged QT interval. A serum calcium level requested now for the first time was 5.7 milligrams per cent. Serum phosphorus was 8.2 milligrams per cent. A positive Chvostec sign was observed; however, the Trousseau sign could not be elicited. She was treated with calcium gluconate. A subsequent visit to the Sinai clinic revealed a serum calcium level of 9.0 milligrams per cent and an otherwise satisfactory physical condition. In December 1948 she was examined in the Accident Room of the West Baltimore General Hospital while having a typical tetanic convulsion with carpo-pedal spasm. This responded to intravenous calcium gluconate.

*History of Present Illness:* The pain in her fingers started on or about February 1949, with pain and swelling in the finger joints of her right hand. One week later, the left hand also became involved, while the right improved. In addition, mental changes had been noted by the family for the past 5 years. These comprised a poor memory, "peculiar" answers, repetition of her own utterances, parroting of the words of others, and suggestibility. There was no dizziness, tinnitus, numbness, paresthesia, or hypesthesia. She had occasional diplopia, and stated that she had been unable to stand or sit up without help for the past 5 weeks, but once up she could walk with a shuffling gait. During her convulsions, she had never experienced fecal or urinary incontinence but had now bitten her tongue several times.

*Physical Examination:* Temperature 99.8°F. Pulse 90 per minute. Respirations 20 per minute. Blood pressure 110 millimeters of mercury systolic and 70 millimeters of mercury diastolic.

Examination revealed a well developed, somewhat obese, white female in no apparent distress. She cooperated only fairly well, was none too alert, had a slightly blank stare on her face, and showed echolalia and perseveration in her speech. Her complaints took the form of a whining cry with little change in facial expression. Her speech was slow, but not slurred. The neck showed an old well-healed thyroidectomy scar. Rales were audible in the left upper lobe and in both bases of the lungs. The heart size was at the upper limits of normal with an apical systolic murmur on auscultation. The abdomen revealed a low, well-healed midline scar. The liver and spleen were not palpable, nor could they be percussed below the costal margin. The extremities had normal arterial pulsations and the legs showed no varicosities or edema.

Neurological examination revealed the above mentioned psychiatric disturbances. The pupils were irregular in size but reacted to light and accommodation. The fundal vessels revealed some tortuosity and increase in light streak. The left optic disc appeared a little blurred as compared to the right. No abnormalities of cranial nerve functions were detected. Her responses were unreliable for a sensory examination. The deep tendon reflexes were greatly diminished. She could sit up and walk unaided, but required much encouragement to do so. There was a pill rolling tremor of her right hand. At times she held her hands in "main d'accoucheur" position. The Chvostec sign was positive.

*Laboratory:* The Sulkowitch test for urine calcium was negative. Blood chemistries: On March 11, the day of admission, the alkaline phosphatase was 4.2 Bodansky units, albumin to globulin ratio 4.1 to 3.1, and $CO_2$ combining power 40 volumes per cent. The blood urea nitrogen was 29.9 milligrams per cent, but rose to 87.7 milligrams per cent on March 16, the day of her death. The serum calcium was 5.1 milligrams per cent on March 11, 5.5 milligrams per cent on March 13, and 4.6 milligrams per cent on March 16, while the serum phosphorus was 7.6, 7.6, and 8.6 milligrams per cent respectively on these dates.

The electrocardiogram again showed the prolonged QT interval of hypocalcemia. Roentgenograms of the skull demonstrated a craniotomy defect on the right side, and calcification in the cerebral portion of the brain beneath the parietal bone area. No bony abnormalities were present in the hands and femora. A chest film showed slight enlargement of the heart in transverse position and infiltration in the bases of both lungs.

*Course in the Hospital:* The patient was slightly confused but afebrile. On March 15, penicillin was started. On March 16, her temperature suddenly rose to 103°F. and she was observed to be in a state of circulatory collapse. Intravenous plasma, calcium gluconate, and dihydrotachysterol were now given but did not seem to improve her condition. Calcium therapy was continued, but she remained in shock and coma. She developed violent right-sided twitching and tremors, her head jerked and pulled to the left, she aspirated some fluid during one of the convulsions, and died.

*Autopsy:* The right pupil was slightly larger than the left. Otherwise, external examination revealed only 4 trephine defects in the right parietal region. An electric saw was necessary for cutting the clavicle; the cortex of the bone was densely thickened while the medullary area was very thin. The ribs, however, appeared normal.

The lungs were congested and edematous with a few patches of hemorrhagic infiltration. Many fibrous pleural adhesions were present on the left. No inflammation was found macro- or microscopically. The heart was slightly enlarged. Atheromatous plaques and some calcifications existed in the aorta, and mild arteriolar sclerosis was seen in the arterioles of most of the organs.

The liver was congested. The pancreas was normal as were the adrenals. There were some pelvic adhesions which did not obstruct the intestines. A collapsed descending colon was explained on the basis of hypocalcemia. Evidence of old chronic cystitis was found in the bladder.

There were small calcific deposits on the surface of the capsule of the kidneys. The spleen was congested, with hypertrophy and hyalinization of the vessel walls and some calcium deposits.

Two small thyroid remnants remained on either side. Together they weighed 30 grams and contained colloid cysts and a small central area of calcification. Multiple sections throughout this tissue failed to reveal any remnant of parathyroid tissue.

The brain weighed 1300 grams. There were no significant external lesions. The cerebral hemispheres appeared symmetrical, with a well developed convolutional pattern and sulci of average width. A moderate degree of diffuse symmetrical congestive change was present without evidence of hemorrhage or areas of softening.

On sectioning the ventricles were symmetrical, of normal size and shape, and the differentiation between the gray and white matter was normal. Throughout all of the basilar nuclear structures, "calcific" change of a finely granular type was seen and felt, this appearing on the cut surface as a slightly raised, punctate, glistening, diffuse granularity. These deposits were concentrated in the external and internal capsules, putamen, and caudate nucleus and extended into the centrum semiovale of the frontal and parietal lobes on either side. (See Figures 1 and 2.) No evidence of infiltration was found grossly in the centrum semiovale of the temporal lobes, the cortical gray matter and subcortical white matter, the arcuate fibers and corpus callosum. The granular gritty change continued rostrally bilaterally in the basal nuclei, to and including the heads of the caudate nuclei and extended into the centrum semiovale of each frontal lobe to within 0.1 cms. of the rostrum. Caudally, it continued similarly in the basal nuclei, especially the entire putamen, and the beginning of the thalamus, mainly in the medial and lateral nucleus, with extension into both centra semiovale in the parietal areas. Again there was no gross evidence of temporal lobe extension or cortical involvement. Further caudad about the level of the superior corpus quadrigeminus, the deposits were of greatly decreased intensity. However, a dense, diffuse infiltration with granular material almost obliterating the myelinated architecture, appeared in the brachium pontis on either side, the dentate nuclei, and the white matter of the cerebellum. No gross evidence of involvement was seen in the red nuclei, the substantia nigra, the choroid plexus of the lateral ventricle, or the pineal gland. Sections taken through the midbrain, pons, cerebellum, and medulla at representative sites showed no gross evidence of granular change in the midline structures.

Microscopically, this extreme deposition was seen in the interstitial capillaries and in the media and adventitia of the small and medium-sized arterioles, many of which were pipe stem in appearance. (See Figure 3). The perivascular spaces were considerably widened. In several areas, deposits were present within the media of some of the veins as well. Some interstitial deposits appeared also without regard to any vascular pattern.

These deposits appeared as fine granules which fused into larger calcospherites, and eventually, the latter coalesced into mulberry-shaped masses. (See Figure 3).

The cortical neurones were not damaged and appeared to be present in normal numbers. Many viable ganglion cells were seen in the midst of massive deposits. In a few instances, however, where the deposits were very dense, the material had found its way into the interstices of the gray matter,

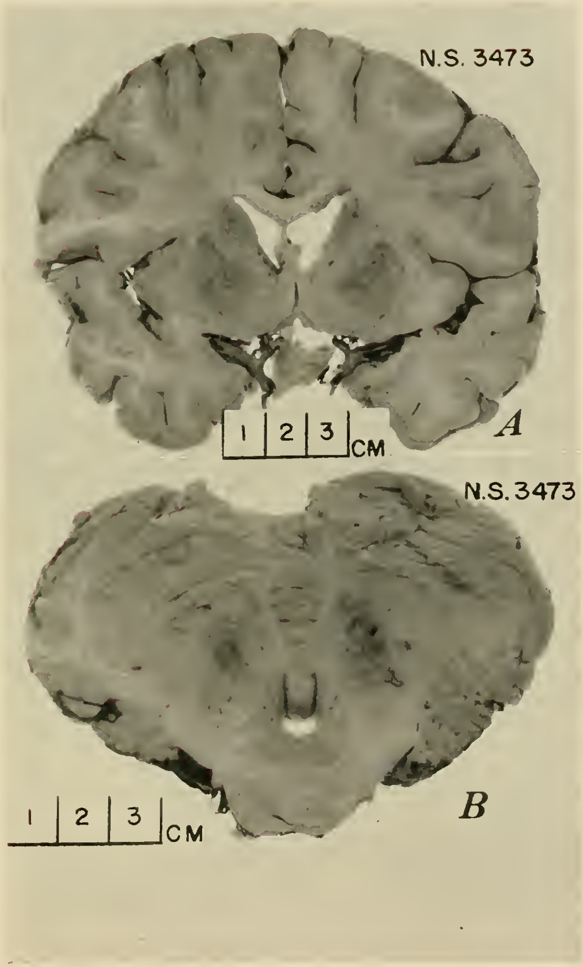

Fig. 1. Photograph of coronal section, showing granular deposit—A. In the basal ganglia extending into the centra semiovale. B. In the dentate nuclei.

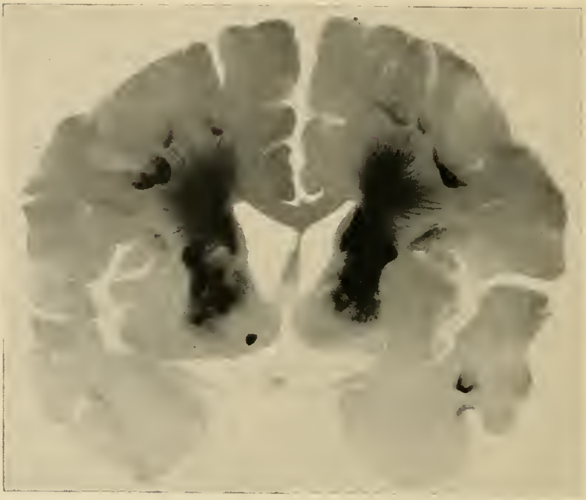

FIG. 2. Roentgenogram of coronal section through cerebrum. Note the fine detail with which the distribution of the calcium deposits can be demonstrated.

being associated apparently with neurone cells and to a lesser degree with macroglia. However, this granular infiltration of neurone cells and macroglia was very minimal and only occasionally seen, the main infiltration being in the vessel walls and perivascular spaces. Myelin sheath stains showed some slight evidence of distintegration of the myelinated pathways. In many instances, however, the myelinated pathways of the internal capsule seemed to course about the masses as though being displaced rather than destroyed by the granular material. The deposits were located in the interstitial tissue of the globus pallidus and putamen rather than in the regions of the myelinated tracts.

Cajal stains revealed no increase in astrocytes in either the gray or white matter of the cortex. However, in some of the more heavily infiltrated areas, astrocytes were increased in number, and in the vicinity of a few of the vessels there was an abundance of astrocytic glia. Some capillary proliferation was evident as well, apparently in an attempt to offset the encroaching change. No inflammatory changes were seen, nor were there any primary vascular lesions. There was thus remarkably little change in the cyto-architecture of both the cerebrum and cerebellum.

Histochemical studies showed the granules to stain dark blue to black with hematoxylin and eosin stains. With thionin, they stained dark blue in most cases, and cresyl violet also stained them dark. With Cajal stains they appeared black. These staining characteristics were found even where the deposits contained no calcium or iron as shown by von Kossa's and Gomori's stains. Masson trichrome stains showed the granules to take the green component, suggesting an organic background. There was no true connective tissue proliferation. The acid fast Kinyoun stain was negative. Von Kossa stains were strongly positive in the area of the basal ganglia, the centrum semiovale, the dentate nucleus, cerebellum, vermis, and brachium pontis. In some of the cortical areas and subcortical white matter, in the midbrain, pons, and medulla, where the deposits were progressively much less dense, they did not stain with von Kossa's stain and hence did not contain calcium. Likewise, at the periphery of the heavy deposits many granules could be seen which contained no calcium. Other granules appeared to be partially calcified, containing calcium salts in their center but not in the

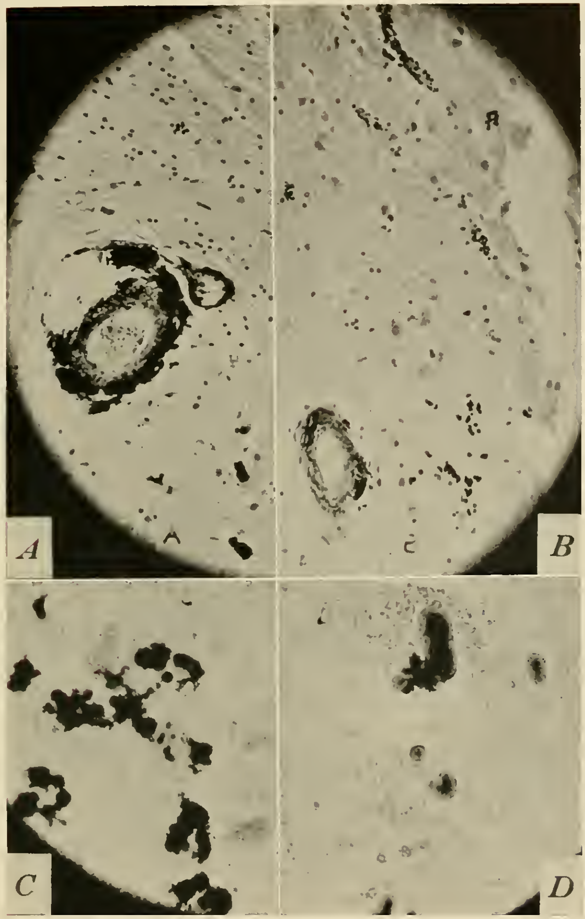

FIG. 3.

A. Massive deposits in wall of both arteriole and vein. Note the widened perivascular space, and the occluded capillaries in the lower right hand corner.

B. Early deposition in arteriolar wall. Note fine granules surrounding the capillaries. Low power. Hematoxylin and eosin stain.

C. Fusion into larger calcospherites and mulberry-shaped concretions.

D. Note calcium in center but not in periphery of concretion. Granules not stained with von Kossa's stain may also beseen. High power. Von Kossa's stain.

periphery. (See Figure 3) Gomori stains were also positive, even in some of the areas where there was no calcium. The iron deposition thus apparently preceded the calcium deposition.

These histological studies thus suggested that the deposits consisted of an organic substance which became subsequently impregnated first with iron and then with calcium salts.

By distribution this calcification of the media of larger and smaller arterioles, capillaries, and some of the veins involved mainly the basal ganglia, external and internal capsules, dentate nucleus and brachium pontis bilaterally. From there it spread to involve the adjacent centrum semiovale. The subcortical white matter and cortical capillaries showed much less extensive involvement. There were some deposits also in the midbrain, pons, and medulla becoming progressively less dense and negative first for calcium and then for iron. Some interstitial granules were found in the granular layer and Purkinje layer of the cerebellum.

The final anatomical diagnosis was: colloid thyroid with pathological calcification (two portions); thyroidectomy, partial (two operations); absence parathyroid glands; cerebral vascular calcification; skull perforations, right parietal, four; pulmonary atelectasis, bilateral; pulmonary edema, bilateral; operative absence of fundus uteri, right fallopian tube, right ovary, and appendix; chronic cystitis.

## DISCUSSION

As noted above, the group of non-arteriosclerotic cerebral vascular calcifications has been termed Fahr's Disease, a diagnosis made on the basis of the histopathology, histochemistry, and distribution of the cerebral lesions. Histologically, small granular deposits are found in the media, adventitia, and perivascular spaces of the small arterioles and venules as well as around the capillaries. These coalesce into larger deposits, forming concentric rings, mulberry-shaped masses, and finally grossly visible stones. No primary lesions can be seen in the vessels themselves. The ganglion cells and white matter only become involved secondarily when the deposits become so massive as to occlude the blood supply. These deposits are found primarily in the basal ganglia and dentate nucleus.

Histochemically, there is described an organic protein substrate which stains dark with hematoxylin, thionin, cresyl violet, etc. before and after decalcification (8). Early small lesions are described as not staining with either von Kossa's or Gomori's stain. As they become older, they stain positive for iron first and then for calcium (9).

Etiologically, this group comprises a wide variety of syndromes; however, of the 63 reported cases many seem to show some endocrine disturbance. Thirty cases had hypoparathyroidism (21, idiopathic hypoparathyroidism; 5, pseudohypoparathyroidism; and 4, postoperative hypoparathyroidism) while 16 more had suggestions of such a deficiency with symptoms of tetany, trembling and contractures of fingers, carpo-pedal spasm, muscular twitches, fibrillations, and cataracts. In 3 of these 16 cases, only 2 parathyroid glands could be found at autopsy. Two of them had hypothyroidism, and 2 more had Addison's Disease.

Of the four reported cases of cerebral vascular calcification following thyroidectomy, two are still alive. In one, a cortical biospy was taken (1), and in the other (2), a presumptive diagnosis was made by history and the presence of bilateral cerebral calcification in the region of the basal ganglia and/or dentate nucleus on skull roentgen films. Of the other two cases, only one author reports the parathyroid glands as absent (3) while the other does not mention them (4).

Hence, only 17 cases remain which may truly be called "idiopathic" non-arteriosclerotic cerebral vascular calcification. If the term Fahr's Disease is to be retained, it is suggested that it be limited to this group of unknown etiology, while considering the larger group of 30 (or possibly 46) as a complication of hypoparathyroidism.

In addition to endocrine deficiencies, most of these patients show epileptic convulsions and mental deterioration or retardation. Since some also have papilledema as well as pyramidal and extrapyramidal signs, it is not surprising that four other patients as well as the one here reported, were subjected to exploratory craniotomy. Hence in cases showing roentgenographic evidence of symmetrical bilateral cerebral calcification, a serum calcium level is extremely valuable in the differential diagnosis (10).

A more extensive review and analysis of these reports will appear in the near future.

## CONCLUSION

A case of bilateral cerebral calcification is reported after two partial thyroidectomies, 40 and 20 years before death respectively. The patient's state of hypoparathyroidism was proved both chemically and anatomically. Histopathologic and histochemical studies showed this to belong to a group of non-arteriosclerotic cerebral vascular calcifications. Its relation to the other reported cases of this group is briefly discussed.

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# CLINICO-PATHOLOGIC CONFERENCE

*From the Case Histories, University Hospital, Baltimore*

## CLINICAL HISTORY

The patient presented here had his appendix removed in 1934 and his tonsils and adenoids, in 1935. Two of his twenty-nine years were spent in southern United States and Europe as a member of the Army.

On the twenty-fourth day of September, 1950, while carrying heavy luggage up a stairway, he suddenly felt weak and dizzy. Sweating profusely, he experienced colicky abdominal pains and an urgent desire to defecate. At stool, a pint of dark red blood and well formed fecal matter were passed. Shortly afterwards, he was admitted to the University Hospital.

For three years prior to this shocking incident, abdominal discomfort described as burning and bloating were endured. Hunger, fatty foods and condiments were unrelated to the severity or frequency of the discomfort. Two years before his current admission, roentgenographic study of his esophagus, stomach, duodenum and gallbladder failed to disclose significant abnormalities. The complaint of this patient continued without modification until 6 months before his last admission. Then mucus appeared in the stool, but there was no blood. A barium enema was performed to no avail. Abdominal discomfort and catarrhal constipation without melena to which he became accustomed continued until he was admitted for intra-alimentary bleeding.

At the time of admission the patient was apprehensive. His skin was pale and of normal texture and warmth. The body temperature was 99 F. A freely movable lipoma was fortuitously found in the left half of the chest wall. The abdomen was of normal contour. There was no rigidity, palpable masses or herniae. Minimal tenderness was experienced when the left lower abdominal quadrant and the umbilical regions were firmly pressed. Normal peristaltic sounds were heard. A scarred Battle incision was present in the right lower quadrant. There were no palpable or visible lesions in the anal canal or rectosigmoid colon.

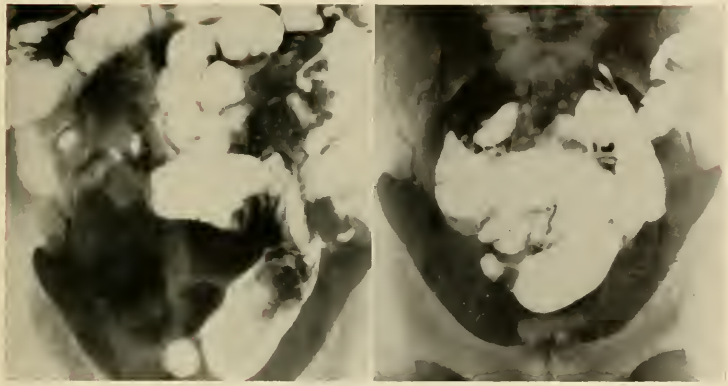
The laboratory reported 23 milligrams of urea per hundred cubic centimeters of blood. There was no evidence of anemia. Cultures of stools and study of blood for agglutinins were uninformative. There were no ova or parasites found.

During the first week of his stay in the hospital the patient had seven bloody stools. Several transfusions notwithstanding, the hemoglobin concentration dropped from 102 to 76 per cent. Five days after admission, the patient suffered gripping abdominal pains, nausea and excessively massive shocking hemorrhages into the gastroenteric canal. He vomited yellowish fluid which in no way was sanguineous. After restorative transfusions, intravenous glucose and saline were given the patient was given a barium enema and oral barium for roentgen ray study of the alimentary canal.

Operative treatment was administered.

## CLINICAL DISCUSSION

*Dr. T. R. Adams:* Prostrating hemorrhage into the gastrointestinal tract unassociated with hematemesis suggests a source in the small or large intestine. Eso-



phageal varices, in the event of rupture, certainly would lead to hematemesis. This patient had none. There was no evidence of liver disease or roentgenographic evidence of esophageal varices. Similarly, peptic ulcer of the stomach or first part of the duodenum can be excluded. Although ulcers of the second portion of the duodenum might cause melena without disgorgement of blood, the roentgenologic studies failed to disclose such a lesion. Then, too, this patient was not dyspeptic. Neoplasms in the upper alimentary tract usually do not cause hemorrhage as dramatic as was seen in this case. The gastrointestinal series help in eliminating these sources. Small bowel tumors, though they bleed, do so in a trifling way and induce obstruction. Terminal ileitis is rarely a cause of massive hemorrhage. The course and symptoms of this patient's illness does not suggest in any way this diagnosis. Neoplasms and diverticula of the large intestine are more likely to cause chronic and not acute massive bleeding. The rectal examination, the sigmoidoscopic and roentgenographic examinations fairly reliably exclude these lesions. Internal hemorrhoids are to be especially remembered as a cause of massive bleeding since one tends to ignore them for more glamorous possibilities. In this manner, I was deceived into doing an exploratory operation for rectal bleeding in a patient who was subsequently cured by a simple hemorrhoidectomy. I hope that this patient does not fall into that category.

In this case, Meckel's diverticulum obtrudes upon my attention. This type of diverticulum can cause obstruction, can be the site of acute inflammation, or the site of peptic ulceration and bleeding or perforation. I expect the anatomic diagnosis to indicate ulcerated Meckel's diverticulum, at least partially lined with gastric mucosa as the cause of this man's hemorrhage.

*Dr. Charles N. Davidson:* In the right lower quadrant there is a loop of bowel that is larger than the others. Protruding from that loop is a small mass of barium. It seems as if there is torsion of this segment of bowel. On later films, the small collection of barium persisted. I thought this was probably a Meckel's diverticulum, but was unable to decide whether it was ulcerated or not.

*Senior Student:* How can you rule out ulcerative colitis?

*Dr. Adams:* I should have mentioned ulcerative colitis. Ulcerative colitis is not, as a rule, a cause of massive hemorrhage. It causes loss of blood over a long period of time.

*Senior Student:* Why wasn't a search made for a diverticulum at the first operation, the appendectomy?

*Dr. Adams:* I suppose the appendix was acute and complicated by localized peritonitis. Under these conditions, one would not explore the small intestine for fear of spreading infection.

*Dr. M. C. Pincoffs:* Dr. Adams, will you comment on cases of massive bleeding, presumably from the large bowel, in older people in which nothing is found to repair, yet they stop bleeding and do not bleed again.

*Dr. Adams:* If a cause of bleeding is not found by diagnostic study methods, the patient should be treated for hemorrhage in the hope that the bleeding will stop. If bleeding persists or alarms by its profuseness, exploration should be undertaken. If the bleeding stops to recur after a time, the diagnostic study should be repeated. I suggest that in cases like this, as a rule one should postpone surgery as long as possible.

## PATHOLOGIC DISCUSSION

*Dr. Hugh R. Spencer:* The gross specimen in this case consists of a portion of ileum measuring 9.5 cm. in length, to which is attached a diverticulum. The diverticulum measures 4 cm. in length and there is an ostium measuring 1.5 cm. in diameter. At the distal end of the diverticulum the mucosa is elevated and somewhat hyperemic, but no definite ulcer is seen.

Section from the diverticulum shows a structure similar to that of small intestine. The distal portion, however, is lined by mucosa which is histologically identical with that of the stomach.

This is considered to be a case of intestinal hemorrhage from a Meckel's diverticulum. Although an ulcer was not found, the fact that bleeding stopped following surgery is presumptive evidence supporting the above diagnosis.

Meckel's diverticulum is a fairly common anomaly of the intestinal tract. It represents the persistence of the proximal portion of the omphalomesenteric duct. In adults the usual position of the diverticulum is approximately 36 inches from the ileocecal valve.

Gastric mucosa, pancreatic tissue, duodenal glands and colonic mucosa represent the various heteropias which may occur in Meckel's diverticulum. Of these, gastric mucosa is the most common. A conservative estimate is that 40 per cent of the diverticula contain gastric mucosa. The peptic ulcers which may develop in these diverticula are the sources of bleeding. In a series of 76 cases of peptic ulceraton in Meckel's diverticulum, 85 per cent occurred in persons under twenty years of age, and 66.7 per cent were in children under ten years of age.

## ANATOMIC DIAGNOSIS

Meckel's diverticulum with heterotopic gastric mucosa.

## MEDICAL SCHOOL SECTION

### ALUMNI CLUB HONORS DR. AND MRS. ELKINS

The University of Maryland Alumni Club of Baltimore was host to President and Mrs. Wilson Homer Elkins at a reception on Thursday evening, October 28 in the Wedgewood Room of the Emerson Hotel in Baltimore.

Dr. Wilson H. Elkins

### DR. WILSON H. ELKINS NAMED PRESIDENT OF UNIVERSITY

Succeeds Dr. H. C. Byrd

Dr. Wilson H. Elkins, formerly President of Texas Western College in El Paso, arrived at College Park September 1 to assume his duties as President of the University of Maryland succeeding Dr. H. C. Byrd who resigned in January, 1954 to campaign for the Governorship of the State of Maryland.

Dr. Elkins, who is 45 years of age, a Rhodes scholar, a member of Phi Beta Kappa and a former star athlete at the University of Texas was selected from a group of over 100 prospects. In a statement to the Baltimore Sun, Judge William P. Cole, Jr., Chairman of the Board of Regents of the University stated that, "We were most impressed by Dr. Elkins' splendid academic training." Dr. Elkins was introduced to many citizens of Maryland over WBAL-TV on the initial opening of the University of Maryland's public service program, TV-MD.

## NEW DEPARTMENT CREATED

### Department of Bacteriology Becomes Department of Microbiology

With the retirement of Dr. Frank W. Hachtel, Professor of Bacteriology, and the appointment of Dr. Charles L. Wisseman, Jr. as Professor of Microbiology, the identity of this department has been changed by the addition of the more inclusive term Microbiology.

## DR. C. L. WISSEMAN APPOINTED PROFESSOR OF MICROBIOLOGY

### Succeeds Dr. Hachtel in Reorganized Department

Dr. Charles Louis Wisseman, Jr. has been recently appointed Professor of Microbiology at the School of Medicine and assumed his professorial duties as of July 1, 1954. Dr. Wisseman replaces Dr. Frank W. Hachtel who is now Emeritus Professor of Bacteriology.

The new Professor is a native of Texas and received his pre-medical education at the Southern Methodist University. After graduation he continued his graduate work in parasitology, receiving the Master of Science degree from Kansas State College in 1942. In 1943 he entered Southwestern Medical College being graduated in 1946. He

Dr. C. L. Wisseman

then entered the United States Army, serving on active duty with the Medical Field Service School at Fort Sam Houston, Texas and the Army Medical Service Graduate School, Department of Virus and Rickettsial Diseases through 1948. He previously served as an intern and assistant resident in medicine at the Massachusetts Memorial Hospital in Boston following his graduation.

Since 1950 Dr. Wisseman has been Instructor in Medicine at the Georgetown University School of Medicine and Acting Director of the Georgetown University Serology and Bacteriology Laboratory. He has also served as an Assistant in the Rickettsial Section of the Department of Virus and Rickettsial Diseases of the Army Medical Service Graduate School, being promoted to Assistant Chief of Medical Affairs in this department in 1953.

Dr. Wisseman's interests have centered chiefly in the field of virus and rickettsial diseases, his publications numbering some 21 original contributions, many in collaboration with Drs. Smadel and Woodward.

Dr. Wisseman is a member of Sigma Xi, Alpha Omega Alpha, the American Society for the Advancement of Science, the American Society of Tropical Medicine and Hygiene, the New York Academy of Sciences, the Society of American Bacteriologists, and the American Association of Immunologists.

Dr. Wisseman will continue his research activities in the field of virus and rickettsial diseases.

## SCHOOL OF MEDICINE PARTICIPATES IN WORLD CONGRESS OF CARDIOLOGY

### Department of Cardiology Stages Closed Circuit Televison Demonstration

Following the official meetings of the World Congress of Cardiology and the 27th Annual Scientific Session of the Maryland Heart Association on September 12–17, 1954, a group of distinguished cardiologists visited the School of Medicine. On September 20 the Department of Cardiology presented a series of films and television demonstrations before this group on certain aspects of cardiology. The complete program is reproduced below.

### MORNING SESSION

I. Cardiac Catherization Angiocardiography. Dr. R. A. Cowley, Department of Surgery, Division of Thoracic Surgery. Dr. Leonard Scherlis, Department of Medicine, Division of Cardiology.

II. Determination of Circulation Time and Blood Volume. Dr. Robert Moss, Department of Medicine. Dr. Joseph Workman, Department of Medicine, Division of Isotopes.

III. Vectorcardiography. Dr. Leonard Scherlis, Department of Medicine, Division of Cardiology.

IV. Pulmonary Function. Dr. R. A. Cowley, Department of Surgery, Division of Thoracic Surgery. Dr. Kyle Swisher, Department of Medicine, Division of Cardiology.

V. Demonstration of Pulmonic Stenosis Pre and Post Operative Cases. Dr. Sidney Scherlis, Department of Medicine, Division of Cardiology.

VI. Motion Pictures. (a) Thrombosis of Abdominal Aorta. (b) Cardiac Surgery. These films were alternated during the morning.

### AFTERNOON SESSION

Television used to demonstrate its effectiveness in teaching

I. Presentation of Case A, by members of the Cardiology Division. Followed by televised operation. Dr. R. A. Cowley, Department of Surgery, Division of Thoracic Surgery.
II. Presentation of a case with data and live demonstration of vectorcardiography, and heart sounds, by television and sound broadcast.
III. Report of work in progress in regard to certain aspects of the pathogenesis and treatment of angina pectoris. Dr. Sidney Scherlis, Department of Medicine, Division of Cardiology.
IV. Television visit to the surgery during a cardiac operation.
V. Experiences in treatment of subacute bacterial endocarditis. Dr. Theodore Woodward, Professor of Medicine, University of Maryland Medical School.
VI. Television visit to the surgery.

## FACULTY INTRODUCED TO DIRECTOR OF MEDICAL EDUCATION AND RESEARCH

Dr. William S. Stone, recently appointed as Director of Medical Education and Research at the School of Medicine, was recently the honored guest at a reception given by the professional schools of the Baltimore Division of the University of Maryland on Wednesday, June 23, 1954 in the South Lounge of the Psychiatric Institute.

Dr. Stone assumed his official duties at the School and University Hospital on July 1.

## MEDICAL LIBRARY NOTES

Between May 1 and August 1, 1954, the following friends presented books and journals to the library:

Dr. R. E. Bauer
Dr. R. M. N. Crosby
Dr. F. W. Hachtel
Dr. E. H. Henrichs
Dr. J. M. Krager
Dr. J. R. Lutz
Dr. R. T. Parker
Dr. M. C. Pincoffs
Dr. M. S. Sacks
Dr. U. T. Slager
Mr. J. F. Strawinski
Dr. H. B. Wylie

The Emerson Drug Company sent to the library a truck load of periodicals in medicine, pharmacy, dentistry, and nursing, from which it was possible to fill in many issues in the libraries' back files.

The present librarian invited Mrs. Ruth Lee Briscoe, Medical Librarian Emeritus, to make a special trip to the library on July 1, 1954. This date was the fortieth anniversary of Mrs. Briscoe's first day as librarian in Davidge Hall.

On Mrs. Briscoe's arrival at the library—forty years later—she was met by the librarian and members of the library staff and asked to pose at the door for a picture to mark the occasion.

Many alumni and other friends of the School of Medicine remember Mrs. Briscoe over those forty years. There is ample evidence of this in such tangible proofs as the Ruth Lee Briscoe Library Fund, established anonymously two years ago by an alumnus and subsequently maintained by further contributions from the original donor and a number of other interested alumni and friends.

It would be a fitting tribute to Mrs. Briscoe's many years of service and of great value to the library to have this fund built into one of permanence. One alumnus here at commencement time sat down at the librarian's desk and wrote a generous check for the fund; and after asking questions about the library's needs sent another even larger check upon his return home. Library-minded alumni who wish to follow suit may use the coupon below.

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To the Librarian, Medical Library, University of Maryland, Lombard & Greene Streets, Baltimore 1, Maryland

Enclosed is my contribution of $______ for the RUTH LEE BRISCOE LIBRARY FUND

Please send me a yearly reminder to help make the fund permanent

MAKE CHECKS PAYABLE TO UNIVERSITY OF MARYLAND BRISCOE LIBRARY FUND

Name ____________________
Address ____________________
____________________

## DEPARTMENT OF PATHOLOGY AFFILIATION

Recently agreements have been formally entered between the Department of Pathology at Lutheran Hospital and the Department of Pathology at the School of Medicine. Under this agreement the pathologic material at the Lutheran Hospital of Maryland becomes available for the under graduate and post graduate teaching at the School of Medicine. Also the facilities of the Department of Pathology at the School of Medicine are being made available to residents and Fellows from the surgical and pathologic services at the Lutheran Hospital of Maryland.

## MEET THE EMERITI

### Dr. Edgar B. Friedenwald

Dr. Edgar B. Friedenwald, emeritus professor of Clinical Pediatrics, is a native son of a famous family in Maryland medical history. The youngest of five sons of Dr. Aaron Friedenwald, he has continued in the tradition and scientific heritage of the medical arts. Born and reared in an atmosphere saturated with medical science, with a father and two elder brothers in the profession constantly before him, his career from the start could have been none other than medicine.

Educated in the Zion Lutheran Parochial School and later at Baltimore City College, he was ready for medical school in the fall of 1899. He naturally chose the school where his father and brothers were on the faculty, the College of Physicians and Surgeons, later consolidated with the University of Maryland. He graduated in

DR. EDGAR FRIEDENWALD

1903, having served a junior internship at Mercy Hospital during his senior year. A portion of this period was spent as a surgical apprenticeship under the late Dr. A. C. Harrison.

After graduation Dr. Friedenwald took a position as a mining surgeon in the coal fields of West Virginia for two years. This was excellent practical experience in those days, and widely practiced in lieu of hospital internship. He returned to Baltimore for a year of postgraduate work in medicine at Johns Hopkins in 1905 and 1906. He returned to Charleston, West Virginia for the next year and a half. During this period he married a charming wife who has since been his constant and devoted helpmate.

Looking to the future and with a yearning for specialization, he took his family to Berlin, Germany, where he studied for one and a half years at Finkelstein's Clinic and at the University of Clinical Pediatrics at the Charite.

Dr. Friedenwald returned to Baltimore in 1911, a full fledged pediatrician. Associating himself with the Pediatrics Department at Mercy Hospital under the late Dr. John Rurah, he began an association with medical students, Mercy Hospital, and clinical pediatrics that continued for more than forty years.

There was a short period of active duty in the Army during the Mexican border conflict in 1916. From this he can recount many interesting experiences.

Retiring from active practice and as head of the Department of Pediatrics at Mercy Hospital in 1951, Dr. Friedenwald maintains an active interest in sports, civic affairs and medicine. He has recently given to the library of the Medical and Chirurgical Faculty his personal collection of almost 200 volumes on the history of pediatrics. This contains many choice and rare editions and will greatly supplement the John Rurah Collection.

Although partly incapacitated by arthritis, Dr. Friedenwald is still a familiar figure about the halls of Mercy Hospital and local medical gatherings. His practical and fatherly advice is welcomed by his contemporaries and all his associates.

Best wishes to Dr. Edgar B. Friedenwald!

### FACULTY PARTICIPATES IN ACADEMY OF GENERAL PRACTICE SEMINAR

The Post Graduate Seminar was held at the Hotel Alexander in Hagerstown, Maryland, on Thursday, May 13, 1954. Members of the School of Medicine participated in the Scientific Program.

Dr. Leonard Scherlis, Associate in Medicine and Dr. R. Adams Cowley, Associate in Surgery, presented a motion picture on "Intracardiac Surgery". Dr. Harry M. Robinson, Jr., Associate Professor of Dermatology and Syphilology, presented a paper on "Cutaneous Manifestation of Internal Diseases". Dr. Lauriston L. Keown, Instructor in Medicine, is currently President of the Maryland Academy of General Practice which sponsored the Seminar. Dr. William T. Layman of the class of 1939 is Secretary-Treasurer.

## CHANGES IN MEDICAL SCHOOL FACULTY

### NEW APPOINTMENTS
### 1954–55

Charles Van Buskirk, Ph.D., M.D., Professor and Head of the Division of Neurology.
Charles Louis Wisseman, Jr., M.D., Professor of Bacteriology and Head of the Department.
Howard B. Bensusan, Ph.D., Assistant Professor of Physiological Research.
Robert P. Boudreau, M.D., Assistant Professor of Radiology.
Otto C. Phillips, M.D., Clinical Assistant Professor of Anaesthesiology.
Paul F. Guerin, M.D., Associate in Legal Medicine.
Janet B. Hardy, M.D., Lecturer in Pediatrics.
Carlton Brinsfield, M.D., Instructor in Anatomy (part time).
Raymond L. Clemmens, M.D., Instructor in Pediatrics and Director Developmental Clinic.
B. Stanley Cohen, M.D., Instructor in Medicine.
Ralph Dunlap, Ph.D., Instructor in Medical Psychology.
D. Carleton Gadusek, M.D., Instructor in Experimental Medicine.
Bernard S. Gordon, M.D., Instructor in Psychiatry.
Richard T. Gross, M.D , Instructor in Otolaryngology and in Ophthalmology.

J. Donald Hubbard, M.D., Instructor in Pathology.
Charles B. Hobelman, M.D., Clinical Instructor in Anaesthesiology.
Lester Kiefer, M.D., Instructor in Pathology.
Frances C. McGrath, Instructor in Psychiatric Social Work.
Herman J. Meisel, M.D., Instructor in Urology.
Benjamin Pope, Ph.D., Instructor in Medical Psychology.
Nathan Schnaper, M.D., Instructor in Psychiatry.
Henry F. Startzman, Jr., M.D., Instructor in Radiology.
Theodore E. Stacy, M.D., Instructor in Anaesthesiology.
Betsey G. Wooton, M.D., Instructor in Psychiatry.
Margaret S. Wilson, M.S.S., Instructor in Psychiatric Social Work.
Robert K. Arthur, Jr., M.D., Assistant in Obstetrics.
Joseph Bierman, M.D., Assistant in Psychiatry.
E. Ellsworth Cook, Jr., M.D., Assistant in Medicine.
Wilford A. Councill, Jr., M.D., Assistant in Urology.
Leon Donner, M.D., Assistant in Pediatrics.
Martin K. Gorton, M.D., Assistant in Psychiatry.
William H. Grenzer, M.D., Assistant in Medicine.
Thomas D. Haupt, M.A., Assistant in Medical Psychology.
Nathan B. Hyman, M.D., Assistant in Radiology.
David Josephs, M.D., Assistant in Pediatrics.
David M. Kipnis, M.D., Assistant in Medicine.
Norman Levin, M.D., Assistant in Obstetrics and Gynecology.
William D. Lynn, M.D., Assistant in Surgery.
Raymond L. Markley, M.D., Assistant in Gynecology.
George A. Maxwell, M.D., Assistant in Gynecology.
Kathleen McGardy, M.D., Assistant in Pediatrics.
Donald J. Roop, M.D., Assistant in Medicine.
Henry Seidel, M.D., Assistant in Pediatrics.
Frederick L. Stichel, Jr., M.D., Assistant in Otolaryngology.
Roger S. Waterman, M.D., Assistant in Psychiatry.
Harold H. Weinberg, M.D., Assistant in Otolaryngology.
David R. Will, M.D., Assistant in Surgery.
Grace L. Finesinger, Ph.D., Research Associate in Psychiatry.
Harry A. Robinson, Ph.D., Research Associate in Psychiatry.
Charles Bagley, III, M.D., Fellow in Psychiatry.
Henrique B. deMoraes, M.D., Fellow in Plastic Surgery.
Donald Helinski, B.S., Fellow in Biochemistry.
Kaneo Hirano, Fellow in Pharmacology (part time).
Walter S. Easterling, M.D., Fellow in Psychiatry.
Leo W. Elgin, M.D., Fellow in Medicine.
Richard L. Glassner, A.B., John F.B. Weaver Fellow in Physiology.
William L. Heimer, M.D., Fellow in Medicine.
Norman A. Hulme, A.M., Fellow in Pharmacology.
Daniel F. Johnston, John F.B. Weaver Fellow in Anatomy.

Albert V. Kanner, Fellow in Psychiatry.
Murray M. Kappelman, B.S., Fellow in Pediatrics.
William E. O'Malley, B.S., Fellow in Pharmacology.
Marvin S. Platt, Summer Fellow in Pediatrics.
Joan Raskin, Fellow in Pediatrics.
Emily M. Rody, Fellow in Psychiatry.
Hirsh Leonard Spegelmann, M.D., Fellow in Surgery, Anticoagulant Clinic.
Robert H. Weaver, B.S., Fellow in Biochemistry.
Arthur Wolpert, Fellow in Pharmacology.
Isadore Tuerk, M.D., Consultant in Psychiatry.
Margaret M. Collins, A.B., Research Assistant in Pediatrics.
Grange S. Coffin, M.D., Research Assistant in Pediatrics.
Fred Davis, M.A., Research Assistant in Psychiatry.
Dorothy DeVantis, Research Assistant in Medicine.
Jose Dobel, Research Assistant in Medicine.
Jorge A. Franco, Research Assistant in Physiology.
Claire K. Heisse, A.B., Research Assistant in Pharmacology.
Julia Neil Hurst, Research Assistant in Pharmacology.
Ruth Hall Johnston, Research Assistant in Psychiatry.
Ann L. Liu, M.S., Research Assistant in Anatomy.
Joan A. Little, A.B., Research Assistant in Gastro-Enterology.
Norma J. Nonken, B.S., Research Assistant in Psychiatry.
Eleanor D. Percy, Research Assistant in Anatomy.
Daniel S. Sax, Research Assistant in Pediatrics.
Vincent J. Speckhart, Research Assistant in Biochemistry.
Anne L. Trucker, A.B., Research Assistant in Psychiatry.
Charles B. Adams, Jr., B.S., Trainee in Cardiology.
Charles F. Carroll, Jr., Trainee National Cancer Institute.
Frank G. Kuene, M.D., Trainee in Metabolism.
Aubry D. Richardson, M.D., Trainee in Cardiology.

PROFESSORSHIPS BY PROMOTION:

John M. Dennis, M.D. From Associate in Radiology to Professor of Radiology and Head of the Department.

Maurice H. Greenhill, M.D. From Associate Professor of Psychiatry to Professor of Psychiatry.

Theodore E. Woodward, M.D. From Associate Professor of Medicine to Professor of Medicine and Head of the Department.

ASSOCIATE PROFESSORSHIPS BY PROMOTION:

Beverley C. Compton, A.B., M.D. From Assistant Professor of Gynecology to Associate Professor of Gynecology.

Ernest I. Cornbrooks, Jr., A.B., M.D. From Assistant Professor of Gynecology to Associate Professor of Gynecology.

Raymond E. Vanderlinde, A.B., M.D. From Assistant Professor of Biological Chemistry to Associate Professor of Biological Chemistry.

ASSISTANT PROFESSORSHIPS BY PROMOTION:

Ruth W. Baldwin, M.D. From Instructor in Pediatrics to Assistant Professor of Pediatrics.

R. Adams Cowley, M.D. From Associate in Thoracic Surgery to Assistant Professor of Thoracic Surgery.

Klaus W. Berblinger, M.D. From Instructor in Psychiatry to Assistant Professor of Psychiatry.

Francis G. Dickey, M.D. From Associate in Medicine to Assistant Professor of Medicine.

William Carl Ebeling, III, B.S., M.D. From Associate in Medicine to Assistant Professor of Medicine.

Lester M. Libo, A.M., Ph.D. From Research Associate in Psychiatry to Assistant Professor of Psychiatry.

H. Patterson Mack, M.D. From Associate in Anatomy to Assistant Professor of Anatomy.

F. K. Morris, A.B., M.D. From Associate in Gynecology to Assistant Professor of Gynecology.

James K. Ramsey, M.D. From Instructor in Pathology to Assistant Professor of Pathology.

Roy B. Turner, B.S., M.D. From Instructor in Pathology to Assistant Professor of Pathology.

Joseph B. Workman, A.B., M.D. From Associate in Medicine to Assistant Professor of Medicine.

## MERCY HOSPITAL NEWS

### VISITING STAFF NEWS

Recently appointed to the Visiting Staff at Mercy Hospital are **Dr. Claude F. Bailey** and **Dr. Wilford A. Councill, Jr.**

**Dr. Charles E. Brambel,** Director of the Anticoagulant Clinic at Mercy, will attend the International Conference on Thrombosis and Embolism, July 20–24, at Basle, Switzerland. Under the direction of Dr. Brambel, and with the technical assistance of Vera A. Capone, a Scientific Exhibit on "Superiority of Vitamin $K_1$ as Counteractant for Coumarin Anticoagulants", will be displayed. Also Dr. Brambel will deliver a paper on "Anticoagulants in Trauma, Gangrenous Conditions and Frostbites."

### RESEARCH FELLOW APPOINTED

**Dr. Hirsch L. Spiegelmann** was appointed Fellow in Anticoagulant Studies on April 1, 1954 for a one year period. He will work under the supervision of Dr. Charles E. Brambel. Dr. Spiegelmann is a graduate of St. Joseph's University, School of Medicine, Beirut, Lebanon, 1946. He trained in Surgery at the Boston City Hospital, Boston, Massachusetts; New York University, Bellevue Medical Center, New York City, and Kings County Hospital, Brooklyn, New York.

## RESIDENT STAFF APPOINTMENTS

Supplementing the list published in the last Bulletin, the following residents have been appointed for 1954–55:

**John O. Sharrett,** M.D., Resident in Neurosurgery

**Robert A. Moore, Jr.,** A.B., M.D., Assistant Resident in Neurosurgery (July 1, 1954–November 1, 1954)

**Israel Howard Weiner,** A.B., M.D., Assistant Resident in Neurosurgery (November 1, 1954–February 28, 1955)

**William Howry Slasman, Jr.,** A.B., M.D., Assistant Resident in Neurosurgery (Mar. 1, 1955–June 30, 1955)

Harry S. Weeks, M.D. has withdrawn his contract as General Practice Intern.

## ROTATING INTERN APPOINTMENTS: July 1, 1954–June 30, 1955

George M. Bauernschub, Jr., B.S., M.D.
Anthony A. Bernardo, B.S., M.D.
Arthur G. Edwards, Jr., B.S., M.D.
O. Norman Forrest, Jr., A.B., M.D.
Daniel H. Framm, B.S., M.D.
Donald A. Gillis, B.S., M.D.
James A. Higgs, Jr., B.S., M.D.
Edward W. Hopf, B.S., M.D.
William P. Houpt, A.B., M.D.
Richard A. Jones, M.D.
Hilbert M. Levine, A.B., M.D.
John F. Murtaugh, Jr., A.B., M.D.
Albert Pats, B.S., M.D.
James H. Teeter, A.B., M.D.
George H. Wall, B.S., M.D.
Alvin J. Walters, M.D.

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# POST GRADUATE COMMITTEE SECTION

## POST GRADUATE COMMITTEE, SCHOOL OF MEDICINE

HOWARD M. BUBERT, M.D., *Chairman and Director*
Elizabeth Carroll, *Executive Secretary*
Post Graduate Office: Room 600
29 South Greene Street
Baltimore 1, Maryland

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By Thomas Orpwood

Mr. Delancy L. Provost, vice-president of the Hearst Corporation, presented a check in the amount of $2500 on behalf of the Hearst Foundation to Dr. T. B. Symons, former acting president of the University of Maryland, in a brief ceremony on Thursday, July 15, 1954. The money will be used to carry on the work for the state-wide promotion of postgraduate education activities by the Postgraduate Education Committee of the University of Maryland's School of Medicine.

The presentation took place on the Baltimore campus and was attended by Leslie H. Peard, Jr., station manager of WBAL and WBAL-TV; Mr. Fred I. Archibald, publisher of the Baltimore News Post; Dr. William S. Stone, Director of Medical Education and Research of the University; Dr. H. Boyd Wylie, Dean of the School of Medicine; Mr. George Buck, Director of University Hospital; Dr. Howard M. Bubert, Chairman and Director of the Postgraduate Education Committee; and Dr. John A. Wagner, Associate Professor of Pathology.

### Order Out of Chaos

Representatives of five medical societies, University of Maryland, Johns Hopkins University, and Doctor's Hospital met Thursday, August 6, 1954, at the Sheraton-Belvedere Hotel in Baltimore for the purpose of bringing order out of chaos in postgraduate education of the medical profession in the State of Maryland.

Made possible through the funds contributed to the Postgraduate Education Committee by the Hearst Foundation, the dinner meeting was called by Dr. Howard M. Bubert, committee chairman, and featured an address by Dr. Douglas Vollan, staff member of the Council on Medical Education and Hospitals of the American Medical Association and an authority of medical postgraduate education.

Dr. Vollan told the group, "There is no end to medical education for the conscientious physician. Instead of a four year education program, education is more likely to be a forty year undertaking."

He went on to say that in a survey, postgraduate education was second only to reading as the best method of continuing medical education after graduation. He declared, "Postgraduate education has already become a regular part of the professional life of many physicians, and as it becomes more widespread, and as it improves in quality there is every indication that more and more physicians will take advantage of such opportunities.

"With the numerous groups engaged in postgraduate education in every part of America, it is to be expected that there is considerable overlap and duplication—if not frank competition—in this field, to the ultimate detriment of all," Dr. Vollan said. He stated further, "Not only is this (overlap and duplication) uneconomical, but the net result is less than optimum quality programs."

"One way to solve the problem," continued Dr. Vollan, "would be to develop a postgraduate institute with the overall objective of promoting, stimulating and coordinating broader and more efficient use of postgraduate education in this area. Basically such an institute could act as a clearing-house of information in this field, and the agency through which long-term objectives to meet the total postgraduate needs of the area could be realized."

The postgraduate authority said in conclusion, "Many of the individual courses offered in this region have been excellent postgraduate programs. But the fact that this meeting has been called is in itself evidence of the recognition of a need for some form of coordinated effort in this field."

The complete text of Dr. Vollan's remarks can be found in the recent issue of the Maryland State Medical Journal, the official publication of The Medical & Chirurgical Faculty of Md.

Following the address a general discussion was held among the representatives. It was the consensus of the group that there was a definite need of a coordinated postgraduate program and it was agreed that a temporary organizing committee would be the step to accomplish the purpose.

Dr. William S. Stone, Director of Medical Education and Research of the University of Maryland, with unanimous approval appointed Dr. Bender B. Kneisley, president of the Medical and Chirurgical Faculty of the State of Maryland, as chairman of a temporary organizing committee to study the coordination of post graduate education in the state, along with Dr. Bubert.

Representatives attending the meeting were Dr. Harvey Stone of the Council on Medical Education and Hospitals of the American Medical Associations; Dr. Theodore E. Woodward, Professor of Medicine and Head of the Department of Medicine, University of Maryland; Dr. George H. Yeager, Professor of Clinical Surgery, University of Maryland, and editor of the Maryland State Medical Journal; Dr. Lauriston L. Keown, President of the Maryland Academy of General Practice; Dr. Conrad Acton, President of the Maryland Academy of Medicine and Surgery, and representing also the Baltimore City Medical Society; Dr. Samuel P. Asper, representing the Johns Hopkins Medical School; Dr. Edwin H. Stewart, Chairman of the Postgraduate Committee of the Medical and Chirurgical Faculty of Maryland; Dr. Nathan Needle of the Maryland Academy of General Practice; and Dr. William Schuman of Doctors Hospital.

The following members of the postgraduate committee of the University of Maryland School of Medicine were also present: Dr. Frank H. J. Figge, Professor of Anatomy; Dr. Louis A. M. Krause, Professor of Clinical Medicine; Dr. Allen Fiske Voshell, Professor of Orthopaedic Surgery; and Dr. John A. Wagner, Associate Professor of Pathology.

## ITEMS

**Dr. Robert E. Bauer,** class of 1946 and Dr. Joseph B. Workman, class of 1946 have announced their association for the practice of internal medicine including radioactive isotopes and hematology. Their offices will be located in the University Hospital in Baltimore, Maryland.

**Dr. Frank P. Dwyer, Jr.,** class of 1948, has announced the opening of his office at the Latrobe Building, Charles and Read Streets in Baltimore for the practice of otolaryngology.

## LUTHERAN HOSPITAL OF MARYLAND AFFILIATES WITH SCHOOL OF MEDICINE

### AGREEMENT FOR DEPARTMENTAL AFFILIATION TO FORM BASIS OF COORDINATED PROFESSIONAL ACTIVITY

The Dean and Faculty of the School of Medicine after negotiation with the Medical Staff and the Board of Directors of the Lutheran Hospital of Maryland (formerly the West Baltimore General Hospital) announced an affiliation agreement which became effective July 30, 1953.

The affiliation agreement is to be effected on an academic plane and at a post graduate level. Ultimately the inclusion of the Lutheran Hospital as a unit in the undergraduate teaching plans of the School of Medicine might become possible.

FIG. 1. Mr. Robert S. Hoyt, Administrator

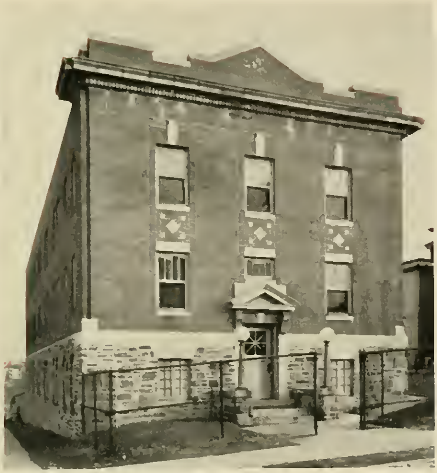

FIG. 2. Interns' Quarters

The unique feature of the affiliation plan is that the master agreement was signed between the School of Medicine and the Hospital as institutions. However, the implementation of the affiliation is to be by departments and through separate agreements instituted and completed under the general agreement plan. The Lutheran Hospital thus joins the group of affiliated institutions closely associated with the School of Medicine.

The Lutheran Hospital started in 1923 as the West Baltimore General Hospital, consisted at first of one building, which was formerly an orphanage. At that time the capacity of the hospital was approximately 125 beds. Gradual improvements, additions and growth have included a 100 bed unit added in 1944 and a new nurses' residence in 1945. The hospital is fully accredited by the American Boards of Surgery, Gynecology and Obstetrics and Internal Medicine. The hospital also operates an approved Nursing School.

In 1950, the Lutheran groups in Baltimore became interested in the hospital and placed a trust fund in its name to be drawn upon at the rate of $70,000 per fiscal year. Additional members were then elected to the hospital's Board of Directors from the Lutheran groups and the hospital was then given its present name, The Lutheran Hospital of Maryland, Incorporated.

This hospital offers approved residencies in medicine, surgery, obstetrics and gynecology and rotating internships. It also offers a diversified, thorough and intense post graduate educational program administered by a director of house officer training.

The hospital is approved by the American College of Surgeons, is a member of the American Hospital Association, the American Protestant Hospital Association, the

FIG. 3. Joint meeting of Attending Staff with Board of Directors. Standing—Dr. Charles Reigner, Member, Board of Directors and donor of Staff Library seen in illustration.

Maryland-District of Columbia-Delaware Hospital Association, the Baltimore Hospital Conference and is a charter member of the Maryland Hospital Services.

The Medical Association of the Lutheran Hospital is among many of the active lay and medical groups interested in the welfare of the hospital. This group sponsors an annual Medical and Surgical Symposium at which nationally known speakers are presented.

The present plant of the Lutheran Hospital consists of 187 adult beds and 35 bassinets. The admission rates include 7,000 adult patients and approximately 2400 deliveries. It has an active accident room serving approximately 10,000 patients annually. The outpatient department is growing rapidly and serves in excess of 7,000 patients.

One of the interesting features of the Lutheran Hospital is its inclusive rate system. The patient pays a single fee for all hospital and allied services, no matter what the extent of the illness or the demand for such services. An open staff hospital, the combined attending and courtesy staffs number over 460 physicians. The staff is organized with an open policy with a well coordinated and functioning organization between the staff committees and those of the Board of Directors.

Following several meetings of the joint affiliation committee the following contract was approved jointly by the Board of Directors of the Lutheran Hospital and the School of Medicine:

1. Under the affiliation, the chiefs of the affiliated clinical departments at Lutheran Hospital are recommended to be included in the Faculty of the University of Mary-

FIG. 5. Attending Staff, Lutheran Hospital 1953

L. to R. 1st row: Drs. Scharfatz, Matchar, L. Ashman, M. Wilder, Krause, Greenwald, Gilmore and Roman

2nd row: Mostwill, M. Levin, Lerner, Schenker, Alessi, Wagner and C. Inman, Sr.

3rd row: Grumbine, E. Wilder, Steinbeck, Welfeld, Wall, Rogers, Checket and Robbins

land, with minimum rank of Associate Professor. The appointment of the chiefs of these services is to apply specifically at the Lutheran Hospital and such appointment need entail no actual responsibility nor necessarily confer any privileges at the University of Maryland Medical School or Hospital.

2. Subsequent appointments as chiefs of departments shall be made by the Lutheran Hospital upon the recommendation of the Dean of the School of Medicine of the University of Maryland. In each instance the Dean shall be guided by the advice of a joint and equal committee appointed by the Dean and the Board of Directors of Lutheran Hospital. From time to time the Chiefs of Departments at the Lutheran Hospital of Maryland may recommend their junior staff men for academic appointment on the Faculty of the School of Medicine, University of Maryland. These appointments in the representative departments will be subject to the approval of the department heads involved and subsequent confirmation by the Dean of the School of Medicine and the Board of Regents of the University. If the Lutheran Hospital appoints a chief of an affiliating department that is not acceptable to the School of Medicine, University of Maryland, that department's affiliation could be automatically dropped by the University of Maryland.

3. Under the proposed agreement, it should be understood that the preclinical and clinical facilities of the University of Maryland Medical School and Hospital will be available to the house staff at Lutheran Hospital for postgraduate study. These facilities will be available under the same conditions that prevail for the house

Fig.

iew

The Lutheran Hospital Professional Scene

1. Operating room
2. X-ray Conference
3. Clinical Laboratory
4. Accident Room
5. Reigner Physician's Library
6. House staff recreation room
7. Staff dining room
8. Delivery Room

staff at the University Hospital. They are arranged for through the Post Graduate Committee.

4. It is further proposed that an exchange of house officers be arranged between the University Hospital and the Lutheran Hospital in the major clinical departments. The periods of such exchange will be agreed upon by the chiefs of the respective departments.

5. The Lutheran Hospital will properly maintain the services of affiliated departments at a standard satisfactory to the University of Maryland, School of Medicine for teaching. They shall also maintain appropriate physical facilities necessary for teaching.

6. As additional physical and clinical facilities are developed at Lutheran Hospital, they will be made available.

7. This contract may be amended by mutual agreement.

8. This agreement is effective as of ————, and may be terminated by either party on two years' notice.

The Administrator of the hospital is Mr. Robert S. Hoyt who was formerly Administrator of the Harford Memorial Hospital in Havre de Grace, Maryland. He is assisted by Mr. Robert Stout, Assistant Administrator. Mr. Carl Hauswald, is Chairman of the Board of Directors.

Organization of the attending staff which includes the department heads is as follows.

| | |
|---|---|
| Chief of Surgery | Dr. William E. Gilmore |
| Chief of Medicine | Dr. Louis A. M. Krause |
| Chief of Obstetrics | Dr. Newton Long (on leave) |
| | Dr. Leon Greenwald (acting Chief of Obstetrics) |
| Chief of Gynecology | Dr. William K. Diehl |
| Chief of Anesthesia | Dr. Harry Ashman |
| Chief of Electrocardiography | Dr. Leon Ashman |
| Chief of Dental Surgery | Dr. Conrad Inman, Sr. |
| Chief of Neuro Psychiatry | Dr. Philip Lerner |
| Chief of Pediatrics | Dr. Milton Markowitz |
| Chief of Dermatology | Dr. Harry M. Robinson, Jr. |
| Chief of Orthopedics | Dr. Hary L. Rogers |
| Chief of Roentgenology | Dr. Paul W. Roman |
| Chief of Ophthalmology | Dr. Roy O. Scholz |
| Chief of Clinical Laboratory | Dr. Solomon Sherman |
| Chief of Urology | Dr. Edward Stinson |
| Chief of Neuro Surgery | Dr. Raymond K. Thompson |
| Chief of Pathology | Dr. John A. Wagner |

Dr. Person M. Checket is Director of House Staff Training

# ALUMNI ASSOCIATION SECTION

OFFICERS*

Frank J. Geraghty, M.D., President

Vice-Presidents

Harold W. Eliason, M.D.
William B. Long, M.D.
Walter Merkel M.D.

Edwin H. Stewart, Jr., M.D., Secretary
Ernest I. Cornbrooks, M.D., Treasurer
Patrick Phelan, M.D., Assistant Secretary
Minette E. Scott, Executive Secretary
William H. Triplett, M.D., Director

**Board of Directors**

Louis H. Douglass, M.D., Chairman
Frank J. Geraghty, M.D.
Edwin H. Stewart, Jr., M.D.
Ernest I. Cornbrooks, M.D.
Patrick Phelan, M.D.
William H. Triplett, M.D.
George H. Yeager, M.D.
Simon Brager, M.D.
Oliver Lloyd, M.D.

**Nominating Committee**

Arthur Siwinski, M.D., Chairman
Gibson J. Wells, M.D.
Emanuel Schimunek, M.D.
Dexter L. Reimann, M.D.
Edwin S. Muller, M.D.

**Library Committee**

Milton S. Sacks, M.D.

**Representatives to General Alumni Board**

Thurston R. Adams, M.D.
William H. Triplett, M.D.
John A. Wagner, M.D.

**Representatives, Editorial Board Bulletin**

Harry C. Hull, M.D.
Albert E. Goldstein, M.D.
Daniel J. Pessagno, M.D.

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* July 1, 1954 to June 30, 1955

## PRESIDENT'S LETTER

I am writing to call to your attention and to the attention of all the alumni what I think is a critical situation, namely, the urgent need for money of the National Fund for Medical Education. This crisis involves the nation's 79 medical schools and threatens the gains of the past and the prospects of the future.

This organization came into existence in 1949 to stimulate private financial support for the medical schools. It was established upon the unanimous recommendation of the University Presidents, the Association of American Medical Colleges and the American Medical Association. The object: a practical solution to the financial deficits facing all of the schools and the establishment of a central national fund to avoid duplication, overlapping and waste of 79 separate fund raising campaigns.

The aims of the National Fund for Medical Education are: to interpret the needs of medical education to the American public, to encourage the advancement of medical training standards in the United States, to preserve academic freedom in the medical schools and equality of educational opportunity and to foster the training of a sufficient number of competent physicians to meet the nation's health needs.

Medical education is a nation-wide responsibility and can be best met on a nation-wide basis. The medical schools of the country graduate physicians who are the nucleus of the country's health resources and who serve all sections of the country. The location of these schools is not geographic but every family, community, and business enterprise, from the big city to the small town, depends on them. Already the shortage of physicians is serious and the demand for increased numbers continues to rise. Medical advances that require competent doctors can no longer be carried out in public health, private practice or industrial medicine unless the medical schools are free of mounting financial deficits. Such deficits prohibit the maintenance of adequate facilities to meet the nation's health needs.

The cost of training a doctor is high: $10,000 to $12,000. Teaching budgets, administration and plant operation have all increased and tuition fees pay only one-fifth of these costs. Endowment income, gifts and annuity funds no longer provide sufficient revenue to meet current operating budgets. Medical schools take up to 40 per cent of the total university budgets with an enrollment that averages only 10 per cent of the total. From this it can be seen that income from other sources must be obtained lest irreparable harm be done to both the medical schools and the universities.

Unless private support is obtained three alternatives are open: lower standards, close down or accept Federal aid and the latter will eventuate in government control. The first two are impossible. Private support appears the only solution for the maintenance of unhampered academic freedom and the independence of the schools.

All funds received are allocated annually to the nation's 79 medical schools for unrestricted use in support of their teaching budgets. The awards are based upon the recommendations of a Grants Advisory Committee representing the Association of American Medical Colleges, the American Medical Association and the Fund's Advisory Council. In 1953 the University of Maryland Medical School received $25,830, of which the alumni contributed $2,690. This year it has received $28,090.50 of which the alumni contributed $3,373.50. This is small in comparison with the amounts contributed by alumni of other medical schools, and I feel sure, will be increased.

Ten million dollars is required annually to support medical education. This must be obtained from the medical profession, public spirited citizens and industry. The medical schools have contributed greatly to industry in the development of full potential for industrial medicine and also the personnel required to staff industrial health programs.

Not only should each alumnus contribute but those engaged in industrial medicine can bring to the attention of business executives the worthiness of the cause and its practical value to industry. Contributions can be made to the National Fund for Medical Education or directly to the respective medical schools. The Foundation recognizes and records these contributions to medical education and lists the names of the donors. Contributions are exempt from Federal income tax.

F. J. Geraghty, M.D., President.

## DISTINGUISHED ALUMNUS CELEBRATES 100th BIRTHDAY

### Dr. John Dawson Sturgeon, Sr., Becomes School of Medicine's First Living Centenarian

Dr. John Dawson Sturgeon, Sr., class of P & S 1880, celebrated his 100th birthday on July 12, 1954, completing over 100 years of association of the name Sturgeon with the practice of medicine in Fayette County, Pennsylvania.

Dr. Sturgeon began the practice of medicine in Fayette County, Pennsylvania in 1881, his father having practiced in Uniontown, Pennsylvania before him (1789–1878). In celebration of his long service to his community the Fayette County Mirror,

DR. JOHN DAWSON STURGEON, SR., Class of 1880, P & S.

a monthly publication of the Fayette County Medical Society dedicated the June, 1954 number to him. Among the many prominent citizens of his community and throughout the country paying him honor and tribute was General George C. Marshall, war time Chief of Staff of the United States Army and formerly Secretary of State. General Marshall in his letter commented as follows—"My first recollection of Dr. Sturgeon was about 60 years ago when I was a heedless, small boy—As a lieutenant on leave I renewed my acquaintance with Dr. Sturgeon which grew in intimacy with the years, though not through frequent contacts.

My mother admired the doctor and often spoke of him. He did me a great service in 1906 which I will never forget. When I saw him last September I was astonished at his apparent well being. He looked well, seemed to feel well and showed fewer signs of age than most men of seventy.

Several months later I spent an evening with King Haakon of Norway. During our conversation he made reference to his age. I did not respond in the usual manner, I think to his surprise, but instead said my views as to age had been greatly altered in the past few months since seeing an old friend, Dr. Sturgeon, of Uniontown, Pennsylvania. Thus I told him of the Doctor's age, his physical appearence, the few lines in his face, and other lack of the usual signs of many years. The King was greatly interested and talked for quite a bit on the subject of Dr. Sturgeon's state of health and physical appearance.

He represents the best of citizenship, a fine example of service to his fellowmen, a model of integrity and simplicity, and a great doctor of the humanities."

The Medical Alumni Association and the Bulletin of the School of Medicine extend to Dr. Sturgeon heartiest greetings as he begins this, the second century of his service in the profession.

## ACCUSATION—REACTION

(*A Letter*)

Baltimore, Md.
October 26, 1954

Dr. J. A. Wagner, Medical Editor
Bulletin of the School of Medicine
Baltimore

Dear Doctor Wagner:

It is believed to be appropriate to offer for public consumption a statement of fact regarding the status of the School of Medicine of the University of Maryland. Our alumni are anxious to know what prompted the tirade against the School of Medicine that has recently been so widely publicized in the newspapers. The Alumni Office has been besieged with inquiries concerning the charges and counter charges that have appeared in the press, consequently this letter is addressed to you hoping you will find it worthy of publication in the Bulletin that all interested may read and thus be advised factually.

All the unfortunate and unfounded publicity given the School of Medicine stems from the so-called exposé by one Mr. Sherwood some three or more weeks ago. It should be noted he is a candidate for an elective office. Apparently he injected his indictment of the School of Medicine into the cauldron of political potpourri with the expectation of gain.

It is unfortunate that fuel was added to the smoldering flame of accusation by the failure of President Elkins and the Chairman of the Board of Regents to take a more positive stand than was initially evidenced by the responses credited to each of them when interviewed and quoted in the newspapers. Their reaction and reported responses, when viewed in the light of numerous inquiries from widely scattered alumni, prompted an investigation by Alumni Headquarters to determine the facts.

Since first things should always come first, contact was made with the agency known to have surveyed and evaluated the School of Medicine in November 1953. That agency was not the Middle States Association of Colleges and Secondary Schools as has been repeatedly reported in the newspapers to have been the case, but the Liaison Committee of the Council on Medical Education and Hospitals of the American Medical Association and the Council of the Association of American Medical Colleges.

The Education number of the Journal of the American Medical Association, wherein will be found listed under the heading "Approved Medical Schools in the

United States," bears the date of September 11, 1954. The evaluation of medical schools as reported therein is based upon the reports and recommendations of the medical surveying agency. Attention is invited to the statement regarding the status of the School of Medicine of the University of Maryland appearing on page 171 of that Education issue of the Journal of the American Medical Association. It will be noted that unqualified approved status has been granted. There is nothing to indicate probation or even probation "in a sense" as has been alleged in the newspapers.

With all this data at hand it is impossible to understand how there could be any question about the status of our School of Medicine. Accordingly, to clear the atmosphere, the Secretary of the Council on Medical Education and Hospitals of the American Medical Association was asked specifically: 1) if that agency had made the survey and evaluation; 2) if the report appearing in the September 11, 1954 issue of the Journal of the American Medical Association was prompted by that survey and evaluation; 3) if that agency had subsequently found any reason to withdraw from its position with respect to approved status for our School of Medicine; and, 4) if that agency had relinquished to another a function in which it was experienced and had been successfully performing for 40 years—that of surveying and evaluating Medical Schools.

His answer to each of these questions was immediate and unqualified. The Council he represented did make the survey and evaluation. It was upon that activity the reported approved status of our School of Medicine appearing on page 171 of the September 11, 1954 issue of the Journal of the American Medical Association was based. There had been no reason to withdraw from the position taken in prompting that report. With respect to the last question posed, intended to correct the impression created by the newspapers that the Middle States Group had denied us accreditation, I quote the Secretary's reply: "The Council has in no way relinquished its long standing function in relation to Medical Schools in the United States to any other agency".

Thus it will be seen that the status of our School of Medicine is not as reported in the newspapers, a fact our alumni will find pleasing. The proof is conclusive, opinion prompted by political controversy to the contrary not withstanding.

It is well known that many improvements in our School of Medicine are needed. Our Dean and Faculty have not been blind to this. Is it not a fact that education is never static—improvement in curriculum and facilities is a growing process in every teaching institution?

Normal growth always requires money and in our case tax dollars are involved. Tax dollars always bring politics and politicians into the picture. I venture the opinion there isn't anything wrong with our School of Medicine a sufficient number of tax dollars won't cure!

What the next survey and evaluation, which it is understood is scheduled for the Fall of 1955, will bring forth remains to be seen.

It has been well established that all depends upon the financial support our Dean, through the Board of Regents, has been pleading for over the years and has been trying to procure through a Legislature, which up to now has failed to understand and appreciate the urgency of our needs. It is possible this unfortunate and unfavorable publicity, born of political expediency, will boomerang and prove to be a blessing in disguise. It could be the means of opening eyes hitherto blind to our financial needs and result in a budgetary allowance sufficient to meet the requirements with which our Dean and Faculty are and have been so familiar. This letter of explanation is not offered defensively. This publicity, unfounded as it has been, has not put our School of Medicine on the defensive. On the contrary, it has forcibly brought to the attention of our alumni in particular and the public in general we are operating a School of Medicine which is approved by the only agency charged with the responsibility of its evaluation. Whether we will merit continued accreditation when again surveyed and evaluated will depend entirely upon the Governor and the Legislature supplying adequate funds. Let it be said it should not be incumbent upon the Dean or any of his Faculty to take time from administrative and teaching pursuits to try and influence any legislator to see the obvious. That is a job which should be undertaken by our alumni. Every interested alumnus, and there should be no exceptions, is needed to man the ship. There is safety in numbers! The things that divide us are trivial when compared with the things that should unite us.

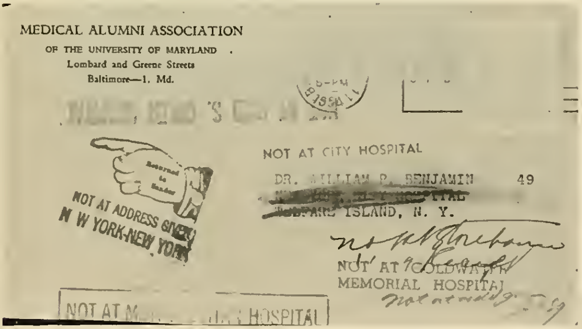
Yours for a better School of Medicine and continued approved status.

Sincerely,
William H. Triplett, M.D., *Director*
*Medical Alumni Association*

## LOST ALUMNI COLUMN

### Alumni Urged to Avoid Joining Lost Alumni Club

Mrs. Minette E. Scott, Executive Secretary of the Medical Alumni Association, reports some 300 to 400 missing alumni whose addresses she cannot obtain. This group of "lost" alumni represents men and women in whom the Alumni Association has a genuine interest and whose names and addresses are most urgently needed. Mrs. Scott reports that she is exerting every effort to locate these missing alumni.



MEDICAL ALUMNI ASSOCIATION
OF THE UNIVERSITY OF MARYLAND
Lombard and Greene Streets
Baltimore—1, Md.

Returned to Sender

NOT AT ADDRESS GIVEN
N W YORK-NEW YORK

NOT AT CITY HOSPITAL

DR. WILLIAM P. BENJAMIN 49
~~[illegible]~~
~~[illegible]~~ ISLAND, N. Y.

NOT AT GOLDWATER
MEMORIAL HOSPITAL

NOT AT M[illegible] HOSPITAL

Lost alumnus Benjamin's letter returns

She requests that all changes of address be reported promptly to her at the Medical Alumni Office, University of Maryland School of Medicine, Lombard and Greene Streets, Baltimore 1 Maryland. Prompt reporting of address changes will avoid a repetition of the envelope herein reproduced. As you see, the forwarding addresses and directory services have been used to locate this alumnus but without avail.

### ALUMNUS-SURGEON ACTIVE AS RADIO AMATEUR

Doctor's Radio Proves Valuable During Flood

An article in the Charleston (West Virginia) Gazette refers to the activities of Dr. William McClung, class of 1940, who is an amateur radio operator—commonly known as a "ham"—on the occasion of a recent flood in Richwood, West Virginia. Dr. McClung's signals went from Richwood to an amateur station at Clarksburg and were relayed from there to Washington where requests for supplies, water purifiers and portable generators were received. He also handled messages for the American Red Cross. Dr. McClung, a practicing surgeon in Richwood, West Virginia, was formerly surgical resident at Mercy Hospital in Baltimore.

## ITEMS

**Dr. James S. O'Hare,** class of 1946, has announced the opening of his offices for the practice of general surgery at 104 West Madison Street, Baltimore, Maryland. Dr. Hare was until recently chief resident surgeon at the Lutheran Hospital of Maryland.

**Dr. August Kiel, Jr.,** class of 1946, has announced the opening of his office for the practice of neurologic surgery at 2 West Read Street, Baltimore 1, Maryland. Dr. Kiel was until recently co-resident in neurologic surgery at the School of Medicine.

**Dr. Nathan Schnaper,** class of 1944, has announced the opening of his office at 1214 North Calvert Street, Baltimore for the practice of psychiatry.

**Dr. John L. Watters,** class of 1952 and Dr. Charles P. Adams, class of 1952 have announced the opening of their offices for the general practice of medicine at 109 Pennsylvania Avenue, Greenville, North Carolina.

**Dr. I. Phillips Frohman,** class of 1937, and a member of the publication committee of the journal "G-P" was a member of a symposium and panel discussion on Medical Writing held in conjunction with the annual meeting of the American Medical Writers' Association in Chicago on September 24. Dr. Frohman spoke on the subject "Medical Writing and General Practitioners."

### CLASS OF 1934 ESTABLISHES STUDENT LOAN FUND

Contributions Are Now Being Received at the School of Medicine

The class of 1934 has recently signified its desire to create a Student Loan Fund. Among those who have expressed intense interest in this project are Dr. Olin C. Moulton of Reno, Nevada and Dr. Wesley J. Ketz of Batesville, Ark. In correspondence recently acknowledged by the Dean, Dr. Moulton and others in the class have signaled the organization of the Loan Fund which will hereafter be known as the "Class of 1934 Loan Fund." In acknowledging the receipt of the fund Dean Wylie signified the gratitude of the School of Medicine for this continuing assistance to medical students.

## DR. JOHNSON HEADS INTERNATIONAL TORCH

At the International Meeting held in Cincinnati, Ohio in May, 1954, Dr. Marius P. Johnson, class of 1930, was elected president of the International Association of Torch Clubs. Dr. Johnson who has also been active in the Baltimore Club will serve as president during the year 1955 when the International Convention will be held in the city of Baltimore.

Dr. Marius P. Johnson

The Association of Torch Clubs founded in 1924 is an inter-professional organization designed to prevent the narrowing tendencies of specialization by developing a breadth of thought and culture and to foster the higher standards of professional ethics and civic well being.

## REGISTRATION, ALUMNI DAY, 1954

*1886*

R. Sumter Griffith

*1895*

Nicholas G. Wilson

*1898*

B. W. Fassett

*1903*

Edgar B. Friedenwald
Clarence W. Lurting

*1904*

Charles Bagley, Jr.
James R. Bishop
Wm. C. Chowning
Edward M. Dailey
Alpheus E. Dann
Edgar B. Davis
Howard V. Dutrow
E. Bruce Eagle
Harlan L. Erwin
Jacob W. Gerber
Andrew C. Gillis
Perry B. Goodwin
J. Henry Orff
Charles L. Parks
Lake Polan
J. Oliver Purvis
R. C. Ralls
Edwin R. Raymaley
Sidney M. Saltz
Norman E. Sartorius
George H. Seaks
J. E. Wilson

*1909*

Harry M. Robinson, Sr.
S. H. Long
C. F. Strosnider

*1911*

F. H. Hutchinson
William H. Triplett

*1912*

Albert E. Goldstein
H. Boyd Wylie

*1913*

Charles Reid Edwards

*1914*

Nolan D. C. Lewis
B. W. Steele
John F. Lutz
R. M. Troxler

*1915*

Frank Shipley

*1916*

Howell I. Hammer

*1918*

Thomas C. Speake
John J. Giesen

*1919*

Raphael Santiago Vasquez

*1920*

Louis C. Dobihal
F. A. Holden
J. Morris Reese
Albert Wild

*1922*

Milton C. Lang

*1923*

Arthur M. Kraut

*1924*

Clewell Howell
Frank J. Theuerkauf
James T. Marsh
A. A. Weinstock

*1925*

Leo T. Brown
M. Paul Byerly
Joseph Nataro

*1926*

H. Edmund Levin

*1927*

T. Nelson Carey
E. Eugene Covington
Byruth K. Lenson-Lambros
Frank K. Morris

*1928*

Hugh Bailey
Simon Brager

*1929*

Hugo Amos
Walter A. Anderson
Henry Bongiorno
Bernard Botsch
Selig Brauer
F. A. Clark
Herman Cohen
Joseph Corsello
W. Paul Dailey
John J. Haney
Joseph F. McGowan
I. P. Morgan
Raymond Sekerak
William J. Sullivan
Henry F. Ullrich
Rafael Vilar-Isern
George H. Yeager

*1930*

Emil Hildenbrand
I. J. Cohen

*1932*

Harry C. Hull
John Edward Savage

*1934*

Thurston R. Adams
D. D. Caples
Robert H. Dreher
John C. Dunbar
John E. Echols
William L. Fearing
Sidney Gelman
Murray J. Hanigsberg
John F. Hartman
William L. Howard
Philip A. Insley
Edward S. Kallins
Reuben Laess
Helen I. Maginnis
Olin C. Moulton
William T. Reardon
Joseph A. Sedlacek
William B. Smith
John N. Snyder
H. Lawrence Sutton
Charles Zurawski

*1935*

Ernest I. Cornbrooks, Jr.
Edward Cotter
Harry M. Robinson, Jr.

*1936*

Harry C. Bowie
D. McClelland Dixon
Walter E. Karfgin
W. Kennedy Waller

*1937*

Eugene Bereston
Everett S. Diggs
Isadore Kaplan
Joseph E. Muse, Jr.
J. King B. E. Seegar
Elton Resnick
Samuel T. Revell

*1938*

Louis C. Gareis
John A. Wagner
Robert C. Sheppard
Theodore E. Woodward

*1939*

Raymond M. Cunningham
C. Hunter Moricle
Leonard Heimoff
Dexter L. Reimann

*1940*

Edmund G. Beacham
James R. Karns
E. O. Daue, Jr.
Arlie Mansberger
Raymond C. V. Robinson

*1941*

John D. Young

*1942*

Joseph Furnari
Theodore Kardash
E. Roderick Shipley

*1943*

Elizabeth Acton
William N. Corpening
Ruth W. Baldwin
D. K. Meade

*1944*

R. Adams Cowley
Champe C. Pool
D. W. Mintzer
Marjorie K. Pool
Paul Tinker

*1945*

John M. Dennis
William A. Holbrook
James H. Shell

*1946*

August Kiel, Jr.
Vernon E. Krahl
Joseph B. Workman

*1947*

Jose G. Valderas

*1949*

Edmund Middleton
Clifford D. Riddel, Jr.
Howard Raskin
Elwin E. Stanfield
John F. Strahan

*1950*

Charles Bagley, III

*1951*

Charles W. McGrady, Jr.
Aubrey D. Richardson

*Faculty*

Monte Edwards
J. Mason Hundley, Jr.
Isador A. Seigel
Ruth Lee Briscoe, Librarian Emeritus

# OBITUARIES

## Dr. Rigdon O. Dees

Dr. Rigdon O. Dees, retired surgeon and physician of Myrtle Beach, South Carolina died in a Conway, South Carolina hospital on July 24, 1954.

Dr. Dees was born in Grantsville, North Carolina on April 14, 1884, the son of the late George and Julia Brinson Dees. A graduate of the class of 1906, he practiced medicine and surgery in Greensboro until June, 1952 at which time he moved to Myrtle Beach. He was a member of the North Carolina Medical Society, the American Medical Association and was active in the research department of the American Cancer Society.

## Dr. Norman S. Dudley

Dr. Norman S. Dudley, senior practicing physician in Queen Anne's County, Maryland and a member of the class of 1901, died at Kent-Queen Anne's Hospital in Chestertown, Maryland on July 1, 1954. Dr. Dudley who was 80 years of age had practiced in Church Hill for over 53 years. His death leaves the town without a physician.

The son of the late Dr. Samuel Charles Dudley he was born in Sudlersville, Maryland and was educated in the public schools of Queen Anne County and was a graduate of Washington College in Chestertown, Maryland. A man of great activity, he at one time was register of wills for Queen Anne's County and served for a time as county health officer. He always maintained an active interest in local sports. In 1953 Washington College honored him at its Fall convocation with a citation as a typical country doctor, emphasizing his more than a half century of medical care to rural Maryland.

## Dr. Jack Fein

Dr. Jack Fein, class of 1932, died at his home in the Bronx, New York, on June 27, 1954. Dr. Fein was 49.

Following his graduation from the School of Medicine he entered the specialty of gynecology practicing at the Bronx Hospital. Early in the Spring of 1954 several hundred of his patients contributed toward a plaque and testimonial attesting to his unselfish service in his community.

## Dr. Erland Harold Hedrick

Dr. Erland Harold Hedrick, class of 1917, a resident and practitioner of Beckley, West Virginia, died at his home on September 20, 1954. Dr. Hedrick was 60. Dr. Hedrick, a former Congressman from the State of West Virginia, had been ill since February, 1953 following a heart attack.

## Dr. Edward Lake Jones

Dr. Edward Lake Jones, class of 1901, died at his home in Kerrville, Texas on July 11, 1954.

A native of East New Market, Maryland, he moved to Kerrville after his retirement about 15 years ago.

## Dr. F. Edwin Knowles, Jr.

**Dr. F. Edwin Knowles, Jr.,** class of 1935, and Acting Professor of Ophthalmology at the School of Medicine, died suddenly at the University Hospital on June 5, 1954.

He was a son of the late Dr. F. E. Knowles of East Orange, New Jersey. After graduating at the University of Maryland at College Park, he completed his studies at the University of Maryland School of Medicine. Dr. Knowles served his rotating interniship at the Mercy Hospital in Baltimore and his residency in ophthalmology at the Baltimore Eye, Ear, Nose and Throat Hospital. For a number of years Dr. Knowles was associated with Dr. Clyde A. Clapp and upon his retirement from the Professorship, succeeded him in an acting capacity. Dr. Knowles was a member of the Mercy Hospital staff, the staff of the Baltimore Eye, Ear, Nose and Throat Hospital and University Hospital and was consulting ophthalmologist at Springfield State Hospital and the Rosewood Training School. He was a member of Nu Sigma Nu and Theta Phi fraternities.

## Dr. Fred W. Rankin

**Dr. Fred W. Rankin,** nominee for the 1954 gold key and Honor Award, eminent surgeon and abdominal specialist, died at his home in Lexington, Kentucky on May 22, 1954. Dr. Rankin was to have attended the annual Alumni exercises at the School of Medicine on June 3 at which time he was to have received the Alumni Honor Award for that year.

A distinguished surgeon and owner of the presidency of three major medical groups, he was the author of over 300 papers on operative and clinical surgery as well as several standard monographs relating to general surgery and diseases of the colon and rectum. A complete biography of Dr. Rankin is contained in the Bulletin of the School of Medicine, January, 1954 (vol. 39, #1).

## Dr. James M. H. Rowland

On Monday, July 26, 1954, Dr. J. M. H. Rowland, former Professor of Obstetrics and former Dean of our medical school, died after a short illness. Born in Cecil County in 1867, Dr. Rowland's early life was characterized by numerous activities, including a short period as a teacher in the public schools of Cecil County. He was a graduate of the Baltimore Medical College in 1892, and immediately became associated with the faculty of this school. For a time he served as a demonstrator in the Department of Anatomy, but soon transferred to obstetrics, a field in which he remained until his retirement as professor in 1937. Dr. Rowland was Professor of Obstetrics at the Baltimore Medical College and shortly after the merger of this school with the University of Maryland in 1913 was appointed to the same post in the new school, resulting from this amalgamation and the addition of the College of Physicians and Surgeons in 1915. In 1916 he was named Dean, School of Medicine of the University of Maryland. He served in this post until 1939, retiring at the age of 73.

In both of these positions Dr. Rowland distinguished himself, proving that he was an excellent administrator and an able representative of the school at national meetings. Under his guidance the school made rapid progress and to him and to his

DR. J. M. H. ROWLAND

efforts may be credited a large amount of our growth and development during the years he was guiding our destinies. However it was as the head of the Department of Obstetrics and as a teacher that he was eminent. Those who were his pupils will always remember the simple yet emphatic manner in which he taught. Many of his sayings and teachings are in use today and have continued because they were correct and were stated in such forceful, simple terms that they could not be improved. For example: at a meeting of obstetricians he read a paper on the teaching of obstetrics at the School of Medicine, University of Maryland. The paper was short and to the point and ended on this note: "We tell them (the students) what to do, we show them how to do it and we make them do it."

He also pioneered in several clinical fields of obstetrics, but received little credit since he was never a prolific writer. He was one of the first to use pituitary extract to induce labor. He had been using amniorrhexis for the same purpose several years before the first paper on this appeared in the literature. And his work in the field of episiotomy was several years ahead of its general acceptance as a procedure of choice.

Dr. Rowland was the recipient of two honorary degrees, D.Sc. and L.L.D. but his innate modesty was such that only a few of his intimate friends knew of these honors. They sat lightly upon his shoulders.

An editorial in The Sun July 29, 1954, three days after his death concludes with these words: "In the long and distinguished history of the University of Maryland's medical school Dr. Rowland is assured recognition among those who have served it faithfully and well."

L. H. Douglass, M.D.

### Dr. Clyde D. Thomas

**Dr. Clyde D. Thomas,** class of 1948 was formerly a member of the house staff of Mercy Hospital in Baltimore. He was currently on active duty with the United States Navy and had an untimely death as the result of an explosion and fire on the Navy Airplane Carrier, Bennington on May 26, 1954.

**Benner, Chandos Miller,** Taneytown, Md.; class of 1905; aged 77; served during World War I; died, April 1, 1954, of coronary occlusion.

**Bizzell, Thomas Malcolm,** Goldsboro, N. C.; class of 1908; aged 68; died, June 9, 1954, of myocardial infarction and hypertension.

**Collier, George Kirby,** Rochester, N. Y.; P & S, class of 1900; aged 75; died, June 18, 1954, of emphysema.

**Covey, William Crockett,** Beckley, W. Va.; class of 1917; aged 61; served during World War I; died, May 19, 1954, of myocardial insufficiency.

**Cramer, Leona Lemon,** Sharon, W. Va.; P & S, class of 1915; aged 63; died, April 24, 1954, of bronchogenic carcinoma.

**Dudley, Norman Spear,** Church Hill, Md.; class of 1901; aged 80; died, July 1, 1954, of cerebral hemorrhage.

**Eley, Lemuel Leslie,** Chuckatuck, Va.; class of 1892; aged 83; died, August 20, 1954, of a cerebrovascular accident.

**Fein, Jack,** New York, N. Y.; class of 1932; aged 48; died, June 28, 1954, of acute coronary occlusion.

**Gerhart, William Francis,** Selinsgrove, Pa.; P & S, class of 1912; served during World War I; aged 66; died, July 2, 1954, of esophageal varices.

**Gordon, Charles Dennis,** Mt. Arlington, N. J.; P & S, class of 1909; aged 76; died, May 14, 1954, of generalized arteriosclerosis.

**Hurtt, Harry,** Still Pond, Md.; class of 1895; aged 81; died, July 25, 1954.

**Johnson, Henry Russell,** Fairmont, W. Va.; class of 1892; aged 88; died, June 22, 1954, of coronary thrombosis.

**Johnson, Walter Brennan,** Cambridge, Md.; class of 1928; aged 52; died, June 4, 1954, of cerebral hemorrhage.

**Anderson, John Reese,** Salt Lake City, Utah; P & S, class of 1915; aged 75; died, March 11, 1954.

**Blalock, Burman K.,** Charlotte, N. C.; class of 1913; aged 67; died, February 25, 1954, of coronary thrombosis.

**Brown, Paul Richard,** Baltimore, Md.; class of 1901; aged 77; served during World War I; died, March 22, 1954, of pneumonia.

**Byrne, John Francis,** St. Paul, Minn.; class of 1910; aged 71; died, January 30, 1954, of coronary pulmonale and pulmonary emphysema.

**Crossett, Homer A.,** Hillsboro, O.; P & S, class of 1914; aged 65; died, December 27, 1953, of coronary occlusion.

**De Rosier, Joseph L.,** Detroit, Mich.; B.M.C., class of 1911; aged 66; died, December 1, 1953, of cerebral hemorrhage with left hemiplegia.

**Skillman, Wilbur Francis,** Baltimore, Md.; class of 1900; aged 75; died, November 6, 1953, of abdominal malignancy.

**Watkins, Daniel Alvey,** Hagerstown, Md.; class of 1903; aged 76; died, September 29, 1953, of cerebral thrombosis and paralysis agitans.

**Willard, Edgar Harold,** Great Cacapon, W. Va.; class of 1908; aged 66.; died, recently of malignancy of the spine.

**Williams, James Marcus,** Warsaw, N. C.; class of 1902; aged 78; died, July 30, 1953, of coronary embolism.

**Hanson, Albert N.,** Santa Monica, Calif.; P & S, class of 1908; aged 82; died, July 18, 1953, of angina pectoris and diabetes mellitus.

**Hearn, Arthur C.,** Baltimore, Md.; class of 1897; aged 79; died, April 19, 1953, of occlusion of the coronary artery and arteriosclerosis.

**Johnstone, Charles William,** DuBois, Pa.; P & S, class of 1906; aged 72; died, July 30, 1953, of carcinoma of the lung.

**Kelly, Bernard V.,** Baltimore, Md.; P & S, class of 1913; aged 64; died, April 14, 1953.

**Knox, John,** Lumberton, N. C.; class of 1906; aged 70; died, June 25, 1953, of acute coronary thrombosis.

**Kolb, Edwin Paul,** Patchogue, N. Y.; class of 1912; aged 69; died, June 14, 1953, of coronary occlusion.

**Kosminsky, Leonce Joel,** Texarkana, Ark.; class of 1906; aged 76; died, May 2, 1953, of acute coronary occlusion.

**Laurent, Francis Victor,** Maitland, Fla.; B.M.C., class of 1908; aged 76; died, July 16, 1953, of bronchopneumonia and carcinoma of the prostate.

**McGinley, Charles,** Lynn, Mass.; B.M.C., class of 1904; aged 74; died, July 29, 1953, of cancer.

**McGregor, Lorenzo Watson,** St. George, Utah; class of 1935; aged 45; died, August 26, 1953, of acute cardiac dilatation.

**Miller, Elmer Manton,** Woodsville, N. H.; B.M.C., class of 1898; aged 80; died, July 1, 1953, of coronary thrombosis.

**Norment, Richard Baxter, Jr.,** Hagerstown, Md.; class of 1914; aged 61; died, April 6, 1953.

**O'Neill, Silverton H.,** Blue Hill, Neb.; P & S, class of 1907; aged 76; died, June 3, 1953.

**Pascall, Thomas Myrick,** Newark, N. J.; B.M.C., class of 1906; aged 72; died, July 17, 1953, of cerebral thrombosis.

**Poole, Albinus,** West Union, W. Va.; class of 1900; aged 84; died, July 21, 1953, of acute leukemia.

**Quinn, Elwood T.,** Jenkintown, Pa.; P & S, class of 1910; aged 65; died, July 28, 1953.

**Robinson, John Ranson,** Van Nuys, Calif.; P & S, class of 1901; aged 84; died, June 22, 1953, of myocardial infarction and arteriosclerotic heart disease.

**Shea, John,** Bridgeport, Conn.; P & S, class of 1911; aged 67; died, June 12, 1953, of acute hemorrhagic pancreatis.

**Sweet, Grover Cleveland,** Rocky Hill, Conn.; P & S, class of 1912; aged 67, died, June 28, 1953, of coronary thrombosis.

**Dill, George Herbert,** Lima, N. Y.; B.M.C., class of 1901; aged 80; died, December 11, 1953, of coronary sclerosis and myocardial infarction.

**Floyd, Albion Elliot,** New Sharon, Me.; P & S, class of 1906; aged 75; died, December 1, 1953, of cerebral hemorrhage.

**Hewitt, John Frank,** Baltimore, Md.; class of 1927; aged 51; died, November 13, 1953, of leukemia.

**Hicks, Calvin Shaw,** Durham, N. C.; class of 1904; aged 74; died, December 19, 1953, of carcinoma of the sigmoid with metastases.

**Keaton, John Cox,** Albany, Ga.; class of 1907; aged 68; died, March 30, 1954.

**Mele, Vincent J., Jr.,** Newark, N. J.; class of 1943; aged 38; died, March 1, 1954, of rheumatic heart disease and uremia.

**Ryan, James Francis,** Providence, R. I.; P & S, class of 1909; aged 72; died, January 28, 1954, of cerebral thrombosis.

**Stiles, Archibald Wilson,** Oswego, New York; B.M.C., class of 1897; aged 81; died, December 23, 1953.

**Udkow, Samuel,** New York, N. Y.; class of 1934; aged 47; served during World War II; died, February 28, 1954, of a heart attack.

**Warren, Robert A.,** Hot Springs, Va.; class of 1907; aged 81; died, December 19, 1953, of myocarditis.

**Weirich, Colin Reid,** Little Chute, Wis.; B.M.C., class of 1901; aged 77; served during World War I; died, March 12, 1954, of congestive cardiac failure.

**Zemp, Ernest R.,** Knoxville, Tenn.; P & S, class of 1894; aged 82; died, February 7, 1954, of acute myocardial infarction.

**Joska, Vincent Vernon,** Baltimore, Md.; class of 1921; aged 55; died, July 1, 1954, of coronary occlusion.

**Kittredge, Patrick James,** Portsmouth, N. H.; B.M.C., class of 1906; aged 77; served during World War I; died, March 20, 1954, of arteriosclerosis.

**Mautner, Emil,** Newark, N. J.; P & S, class of 1903; aged 76; died, April 10, 1954, of a heart attack.

**Pearcy, Charles Lewis,** Salem, W. Va.; P & S, class of 1907; aged 71; died, May 28, 1954.

**Rogers, William Russell,** Bristol, Tenn.; class of 1901; aged 78; at one time Mayor of Bristol, Va.; died, May 1, 1954.

**Shea, Cornelius Joseph,** Jersey City, N. J.; class of 1933; aged 47; served during World War II; died, April 24, 1954, of coronary thrombosis.

**Smith, Houghton Currier,** North Amherst, Mass.; B.M.C., class of 1903; aged 77; died, June 13, 1954, of coronary thrombosis.

**Spoon, Samuel Clarence, Jr.**; class of 1918; aged 58; died, June 2, 1954, of cerebral thrombosis.

**Teal, D. Thompson,** Chesterfield, S. C.; B.M.C., class of 1893; aged 83; died, July 19, 1954, of carcinoma of the prostate.

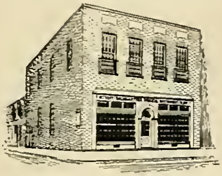
**Thomas, Eugene Marvin,** Prattville, Ala.; P & S, class of 1907; served during World War I; aged 75; died, March 15, 1954, of cerebral hemorrhage.

**Thompson, Earle Xavier,** Piney Point, Md.; P & S, class of 1912; aged 62; died, March 1, 1954, of coronary occlusion and chronic encephalitis.

**Vernaglia, Anthony Paul Joseph,** Hawthorne, N. J.; class of 1928; aged 49; died, June 9, 1954, of cerebral vascular accident.

**Werner, Walter Ignatius,** Albuquerque, N. Mex.; class of 1923; aged 56; died, August 22, 1954, as the result of a plane crash.

**Whaley, Thomas B.,** Princess Anne, Md.; class of 1924; served during World War II; aged 52; died, May 15, 1954, of injuries received in an automobile accident.



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